THE American JOURNAL OF Surgery

NOVEMBER 1954

THURSTON SCOTT WELTON, M.D., EDITOR

Papers of the American Proctologic Society

SIXTY-THIRD YEAR OF PUBLICATION

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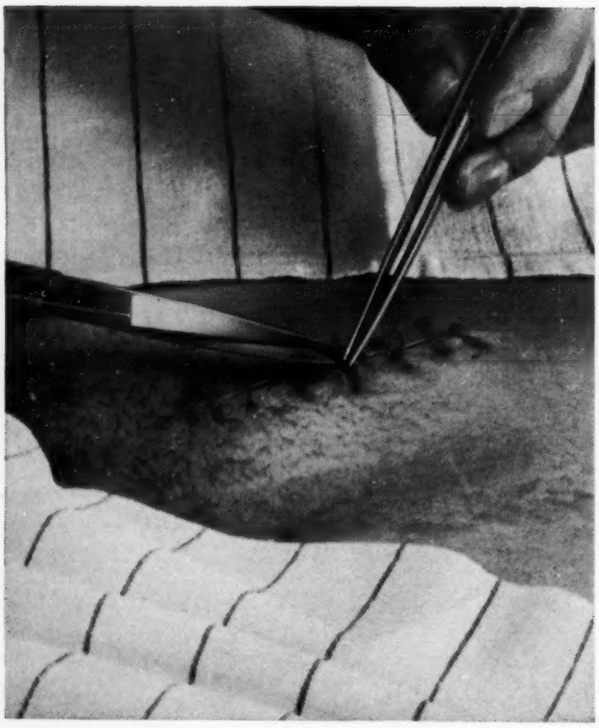

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1. Brown, S., and Fehlman, B. F.: Missouri Med. *50*:609 (Aug.) 1953.
2. Stephen, C. R., Nowill, W. K., and Martin, R.: Anesthesiology *13*:646 (Nov.) 1952.
3. Flowers, C. E., Jr.: Am. J. Obst. & Gynec. *65*:1027 (May) 1953.

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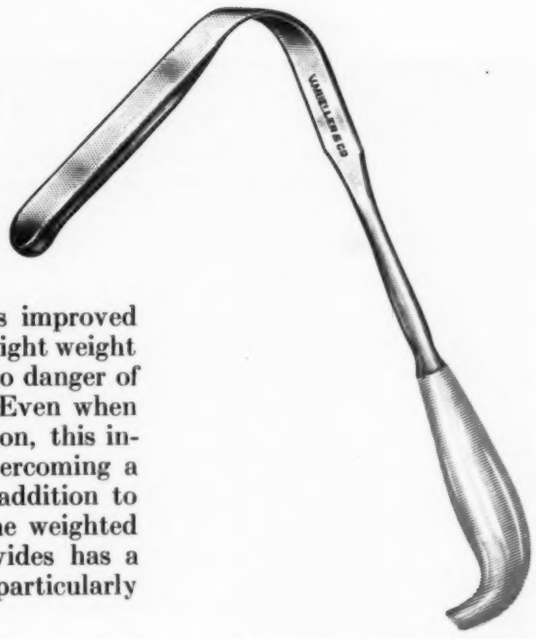

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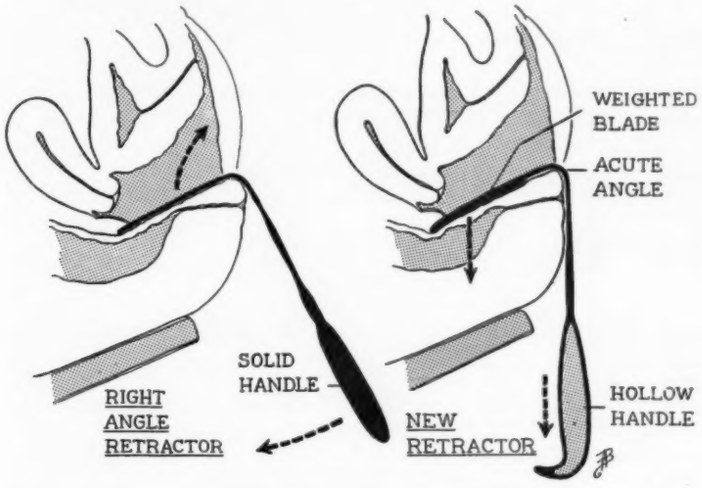

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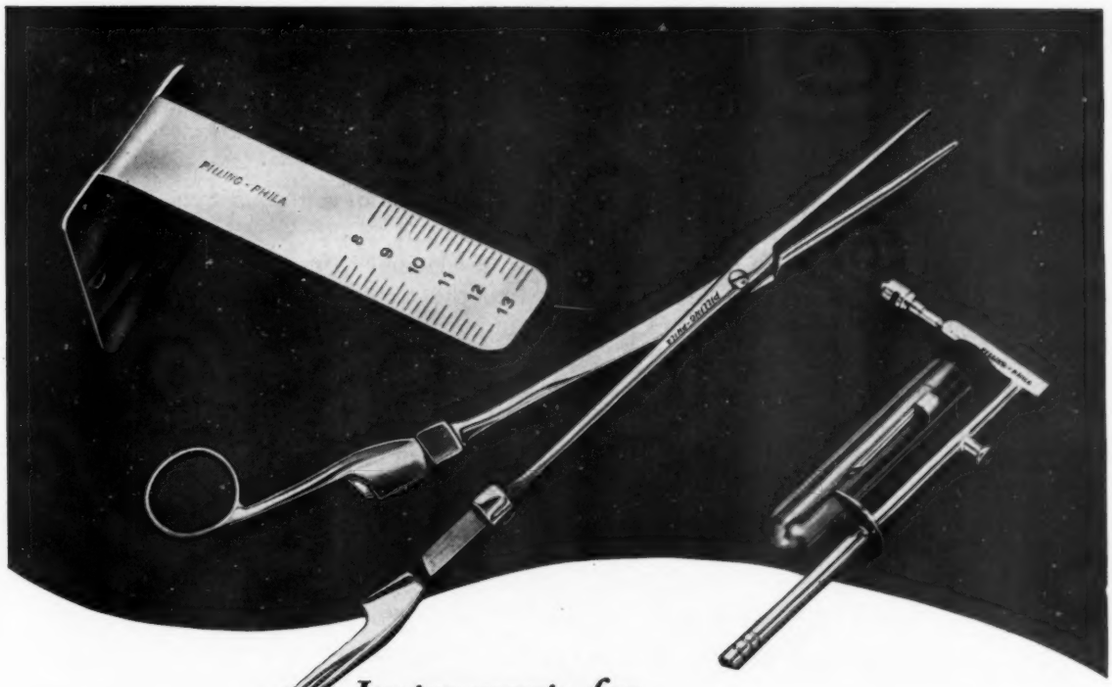

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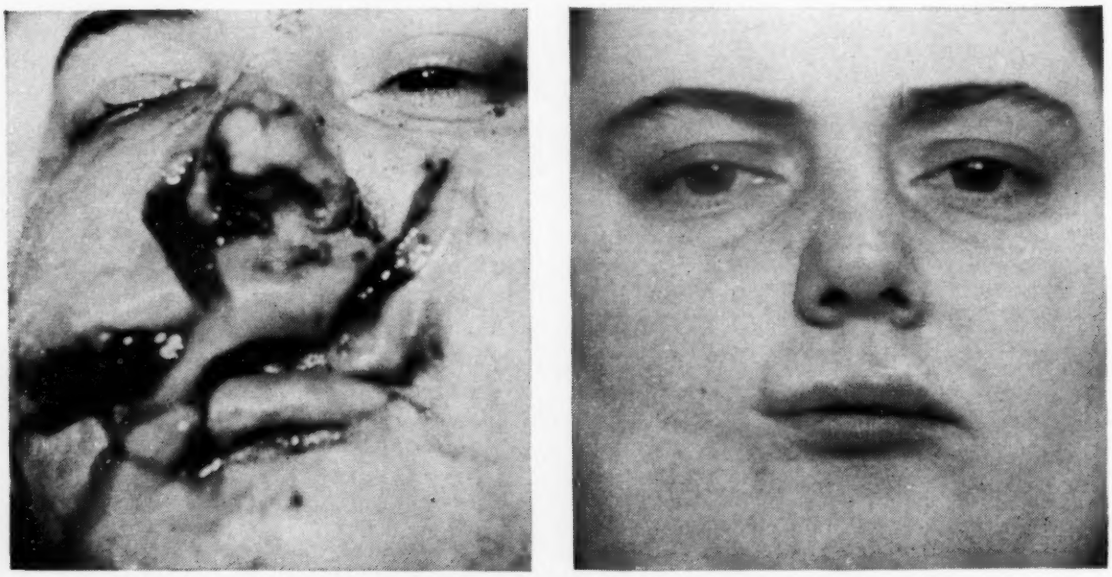

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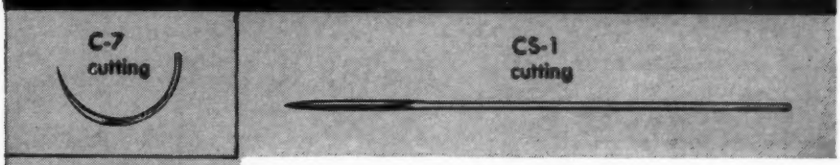

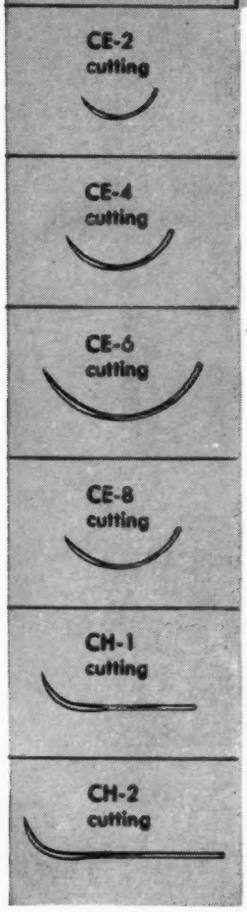

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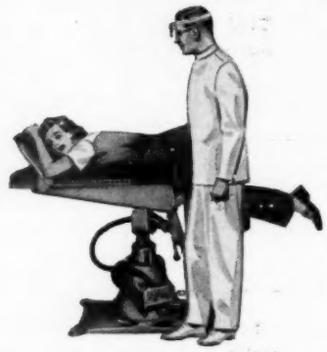

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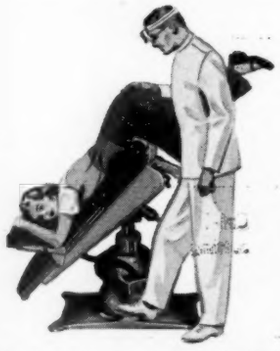

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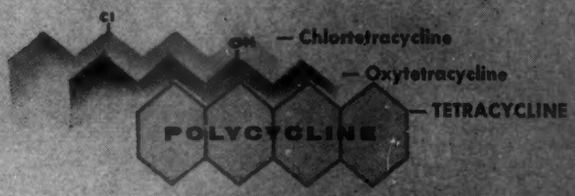

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the only tetracycline produced directly by fermentation from a new species of Streptomyces isolated by Bristol Laboratories...rather than by the chemical modification of older broad-spectrum antibiotics.

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less toxic
(lower incidence of side reactions)
than older broad-spectrum antibiotics.

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Dosage:
average adult,
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children in proportion
to body weight.

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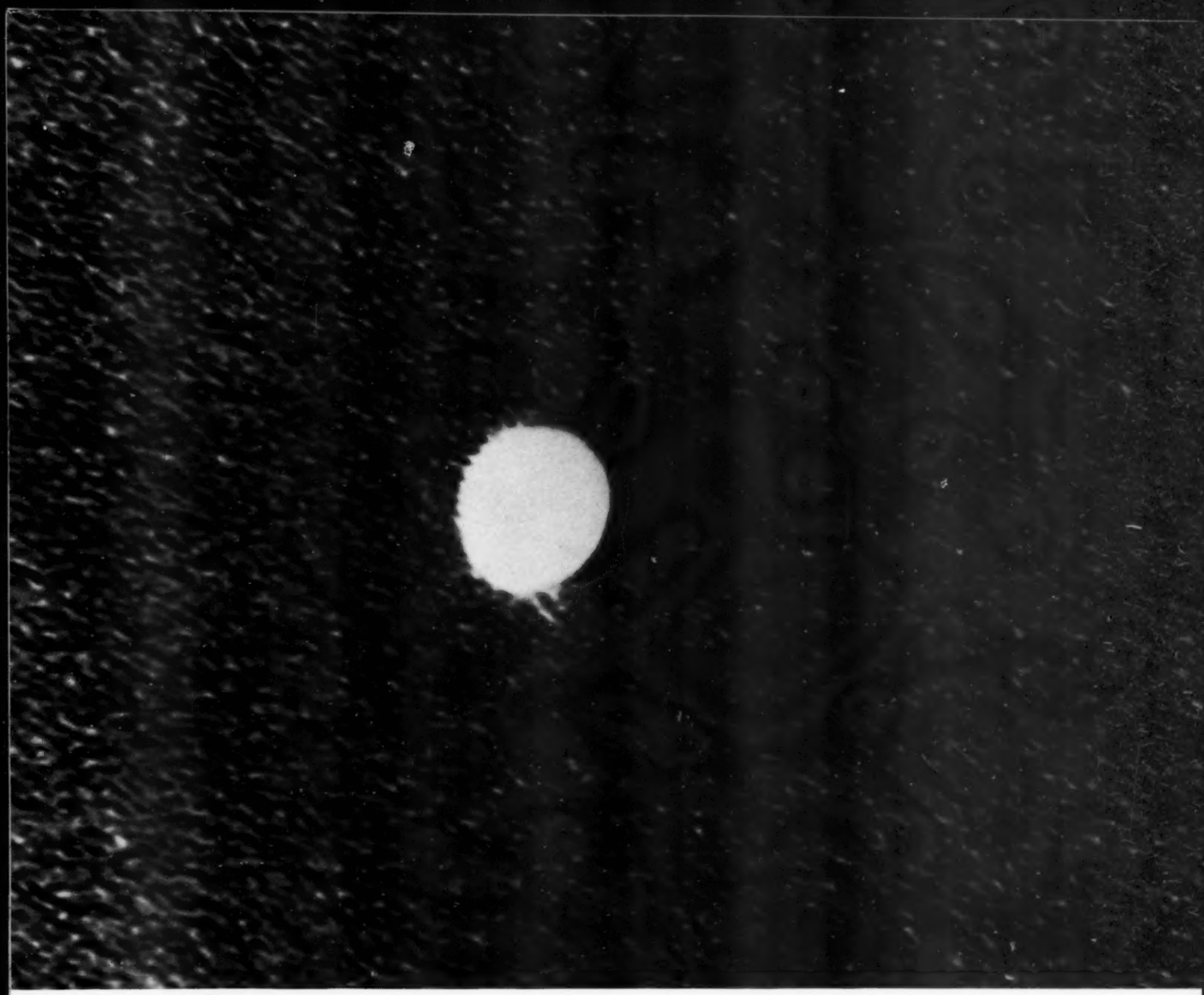

ELECTRON PHOTOMICROGRAPH

Staphylococcus aureus 35,000 X

Staphylococcus aureus (Micrococcus pyogenes var. aureus) is a Gram-positive organism commonly involved in a great variety of pathologic conditions, including

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Rapid onset of action

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Few undesirable side effects

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Effective in low concentration

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REFERENCES:

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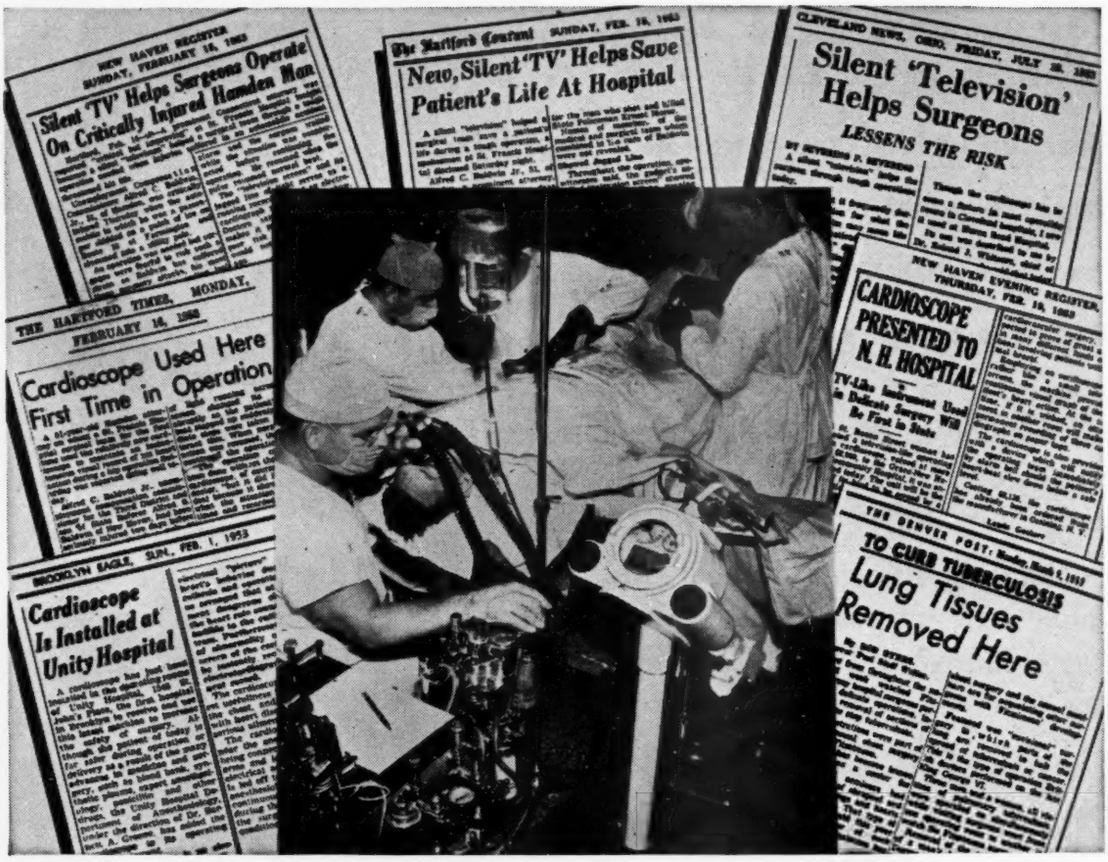
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The Cambridge Operating Room Cardioscope continuously shows the Anesthesiologist and Surgeon any lead of the patient's electrocardiogram and the instantaneous heart rate. Its sweep second hand is most convenient for timing respiration and for similar purposes. A cardiac standstill alarm gives audible warning *instantly* when the heart rate drops below a selected critical minimum.

The Cambridge "Simpli-Scribe" Direct Writing Electrocardiograph is a valuable adjunct to the Cardioscope. By simple operation of a remote control switch on the Cardioscope, any portion of any lead of the electrocardiogram is instantly recorded upon the Direct Writer, which is located outside the operating room without attendance. In this manner, a continuous history of the patient's heart can be written during the entire operation.

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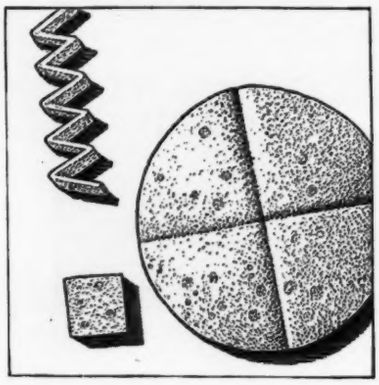

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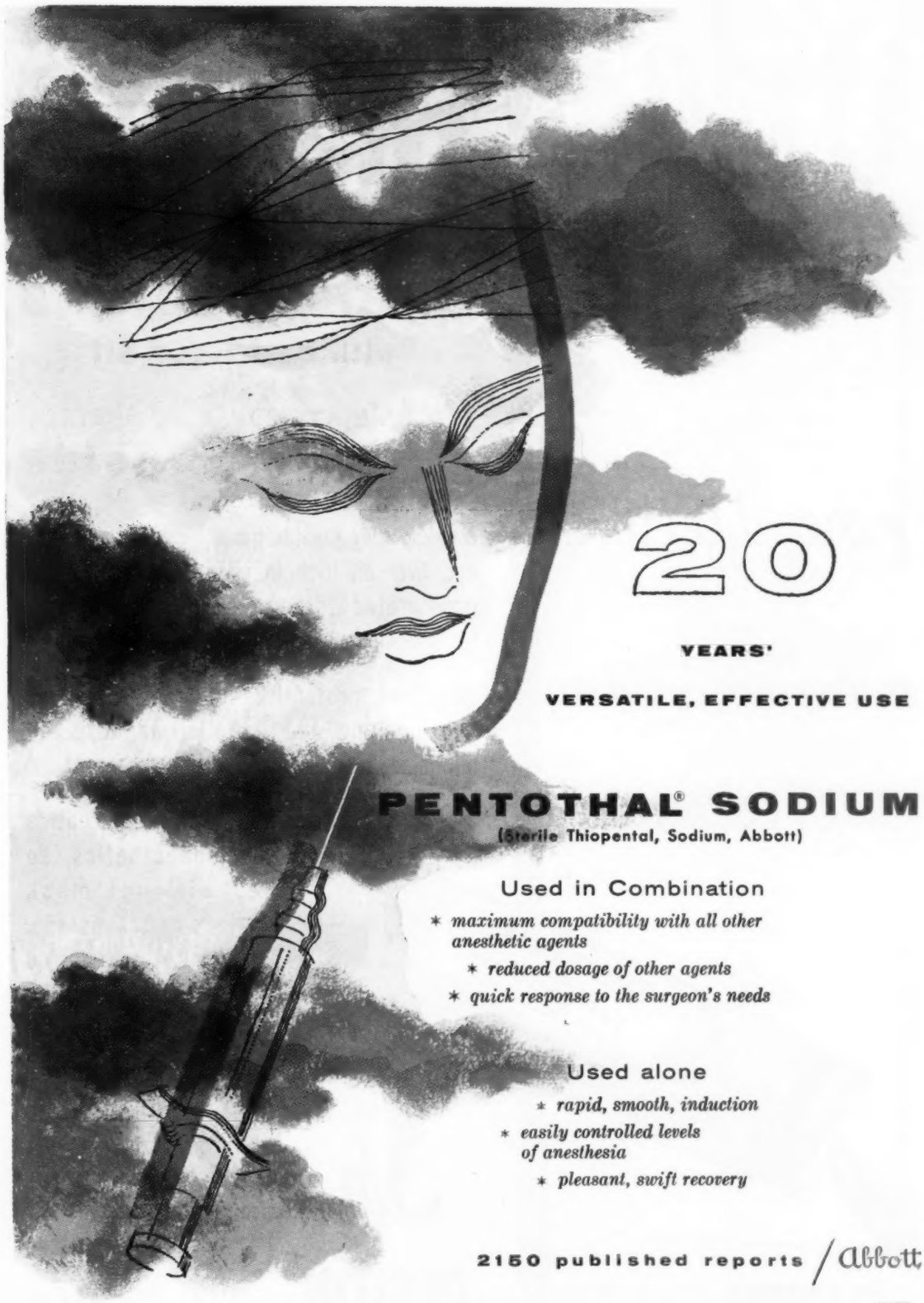
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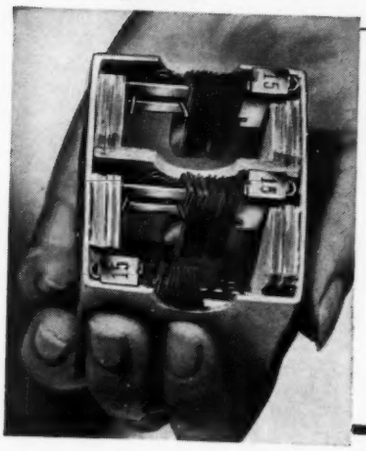

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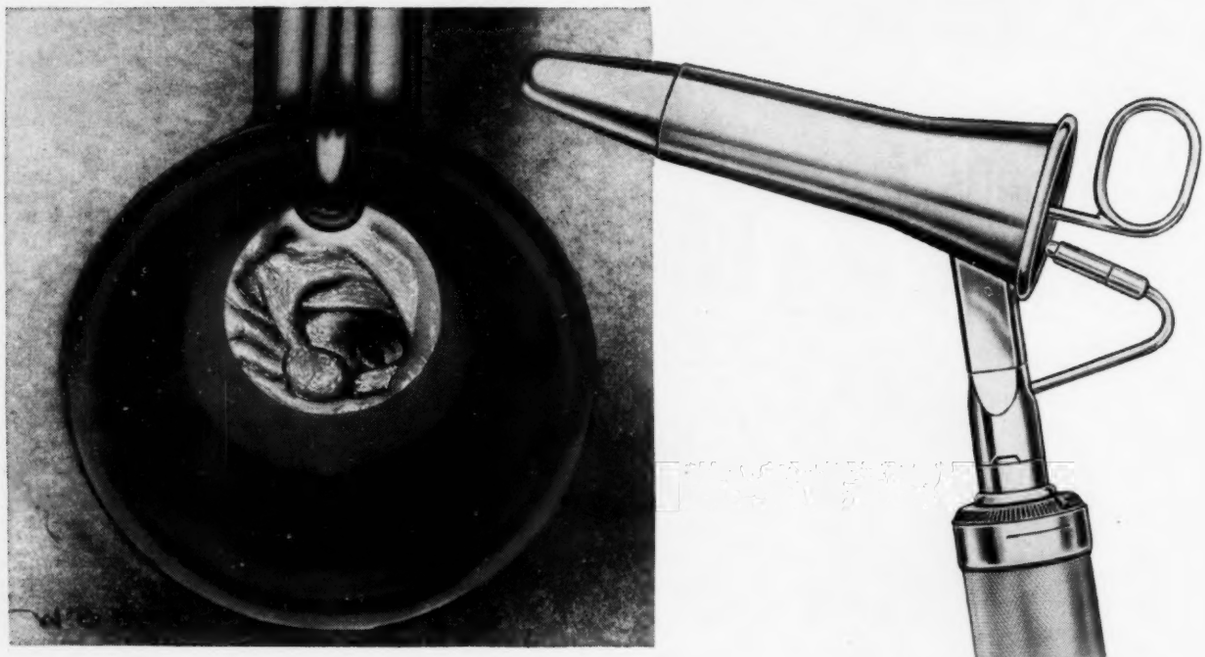

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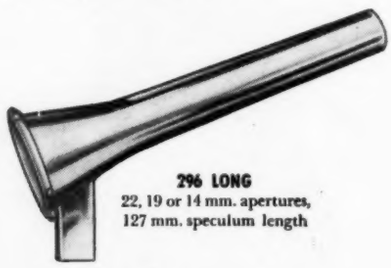

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VOL. 88 NOVEMBER 1954 NUMBER FIVE

Presidential Address

THE flood of criticism that is currently being directed against the practitioners of medicine has caused our profession to reflect upon its shortcomings and engage in some soul-searching of its own. This cannot fail to have a beneficial effect although there is nothing new in this concern of the physician over his own inadequacies and those of his colleagues. In the preface of an early text on proctology published in 1887, the authors voiced their concern over the practices of what they referred to as "the modern Western Rectal Specialist."* They wrote: ". . . he now undertakes to treat other common rectal disorders as well, in a fashion peculiarly his own and suited rather to his own convenience as an itinerant than to his patients' real welfare."

We still have the counterpart of the old "modern Western Rectal Specialist" with us today in all parts of our country in the attenuated form of the self-styled proctologist who directly and by inference claims that he has certain methods of his own with which he can treat all rectal disorders without causing the patient any discomfort or loss of time and without the benefits of hospitalization. Practitioners of this type will be with us as long as gullible patients can be found, and vocal opposition alone will not be sufficient to counteract the discredit that they reflect upon our specialty. We can best oppose the noxious influence of these practitioners by the constructive method of equipping ourselves to provide our patients with the finest scientific care and by establishing a sound physician-patient relationship with those who seek our services. In the remarks that follow I will touch upon what our Society has done and what our individual members can continue to do to meet the criticism that has been directed toward the medical profession and our component specialty.

Proctology as a specialty was established by foresighted physicians who early recognized that the medical rights and privileges of the patients were being violated by the irregular practitioners of that era. This group of charlatans flourished near the turn of the century because of the indifference of the medical profession to the treatment of those who were suffering from disorders of the colon and rectum.

This Society has played a leading role in establishing our specialty upon a sound scientific basis and in bringing to it the dignity and prestige that it now enjoys. The motivating power of this organization has been the strong desire on the part of its members to interest fellow practitioners in the specialty in order that better medical care be available to those afflicted with proctologic disorders. It is for this reason that membership has not been limited to a select group of specialists but has been open to all practitioners who had a genuine interest in preparing themselves to render better care to those of their patients who presented themselves with proctologic complaints. Our Society has encouraged the formation of regional proctologic groups which have no official connection with this organization but serve to stimulate interest in the various

* ANDREWS, E. and ANDREWS, E. W. Rectal and Anal Surgery. Chicago, 1889. W. T. Keener.

phases of our specialty. For many years those in charge of our scientific programs have sought to present a well balanced group of subjects both directly and indirectly related to proctology. An important feature of each annual meeting has been a symposium conducted by physicians well versed in some particular phase of our specialty. This year, for the second time, an additional day has been made a part of our annual meeting so that it could be devoted to a consideration of the basic sciences presented by faculty members of the universities in the city in which we assemble. Largely through the efforts of some of our individual members accredited residencies have been established and maintained to train physicians in methods of proctologic diagnosis and treatment.

It is in ways such as these that our Society has endeavored to fulfill its obligations to our founding members. However, this policy has placed upon us an added responsibility to both our patients and to our profession. To stimulate the interest of our fellow physicians in the diagnosis and treatment of proctologic disorders is not enough; we must maintain the high standards already established and continue to insist that those upon whom membership is conferred meet all the qualifications of the classification to which they are elected.

The American Board of Proctology has been established for the certification of specialists in our particular branch of medicine. The Board is an autonomous entity unassociated with this Society although we have the privilege of electing four of our members to represent us in its deliberations. Certification by an examining Board does not denote the full measure of any physician's medical ability; it is the recognition that he is given by his colleagues that is the true index of his professional stature. However, certification by the Board is tangible evidence that a physician is qualified to render the type of medical care to which the patient is entitled.

Up to this point I have briefly touched upon the creditable manner in which our Society as an organization and proctologists as a group have assumed the responsibility for providing better care for those patients who have turned to us for medical attention. The remainder of the time at my disposal will be devoted to a consideration of the obligations of the individual physician to those who are in need of his services. Many of the following comments would be applicable to any practitioner of medicine but the nature of this Society is such that I will deal with the subject from the standpoint of the proctologist.

It would be impossible to fulfill the obligations placed upon us if we failed to maintain a close physician-patient relationship. This may be difficult at times because we are frequently called upon to serve our patients in the role of consultants. In this capacity we must reveal to each patient our genuine interest in his welfare and we must avoid giving him the impression that we are merely acting as indifferent superspecialists.

The nature of our specialty is such that it can best be practiced within the confines of our office and of our hospitals. This fact, however, does not relieve us of the responsibility of providing medical care to our patients whenever or wherever it is needed. Although we are engaged in the practice of a limited specialty, we are none the less physicians and as such are obligated to provide emergency medical service whenever called upon. Whether we render this service ourselves or through a designated substitute, the responsibility is ours until the emergency has been met.

Unnecessary demands are often made upon the harassed physician by the thoughtlessness of an ill but self-centered patient. Nevertheless, we should never lose sight of the fact that the patient, with his real or fancied illness, is entitled to the consideration which he too frequently fails to show us.

Every patient is entitled to receive whatever time and attention may be necessary to enable us to reach at least a tentative diagnosis. If the time is not available at the initial examination, arrangements can be made for the patient to return at a designated hour when he can be given the attention which he deserves and quite properly expects.

The anatomic limits of our specialty are such that most of the involved areas are accessible for direct examination. This should enable us, with the proper expenditure of time and effort, to reach a correct diagnosis to a degree seldom attained in any other branch of medicine. However, in spite of the restricted field in which we work there can be no justification for us to lose sight of the fact that we are dealing with only one integral part of that marvelous and awe-inspiring organic entity known as the human body. Although we may

carry the designation of specialists in proctology, above all we must remain physicians dedicated to the art and science of healing.

Each patient should be given a reasonable explanation of the results he can expect from the treatment that he is to receive. In periods such as this, when the tensions of daily life are reflected in many of those whom we see in our consulting rooms, it is not enough to control the symptoms with medication and dismiss the patient with the impression that he will have no further difficulty. Patients of this type are in desperate need of a simple explanation of the nature of the functional disorders which are the source of their deep concern. All patients are not capable of accepting such an explanation, but those who do will leave our office better prepared to meet the vicissitudes that they are bound to encounter in subsequent daily life.

If the contemplated surgical operation may result in a permanent or temporary colostomy, it is our responsibility to mention this possibility to the patient. It can then be explained that a colostomy is at times the necessary consequence of a radical surgical procedure and is in no way incompatible with a comfortable and useful mode of life. Those who have followed this practice have seldom encountered a patient who for this reason has denied himself the advantages of a radical surgical procedure. Instead, they have gained for themselves a patient who from the outset has granted his physician his full confidence and complete cooperation. Failure to discuss this subject with the patient prior to an elective surgical procedure is, in my opinion, a violation of his rights as an individual.

No sweeping statement can be made concerning the desirability of discussing with a patient who has carcinoma the exact nature and extent of his illness. This is a matter which must be decided upon an individual basis and is one which calls for a thorough understanding of human nature. However, regardless of the manner in which it is done, we are morally obligated to prepare such a patient to face reality courageously without depriving him of the hope that is essential for his continued existence.

The indiscriminate employment of laboratory and roentgenologic procedures is no substitute for the conscientious medical attention to which every patient is entitled. In evaluating the physical status of a patient, the dependency of some physicians upon the information obtained from laboratory procedures leads one to surmise that they are seeking a method by which they can discard the information gained through the exercise of their native intelligence and rely for their diagnosis upon the results obtained by processing a punched card through a standard I.B.M. machine.

Without the intelligent use of laboratory procedures we would deprive our patients of the scientific tools that are at our command. However, unless utilized with a modicum of common sense, a multitude of laboratory tests can add little to our knowledge of the patient but can contribute greatly to the cost of his medical care.

Consultation service should not be requested without good cause nor should it be withheld whenever deemed necessary. The principle of allowing the patient his free choice of a physician should also be applicable when a consultant is to be summoned.

For the benefit of both the patient and the hospital we should formulate definite plans to coordinate the services that will be required during the time of hospitalization. Laboratory procedures, x-ray studies and consultation service should be planned in logical sequence so that one does not interfere with the other to prolong unnecessarily the period of hospitalization. To inform the patient of the necessity for certain procedures and charges will provide him with a better understanding of the high cost that is so frequently associated with good medical care.

Any patient who displays an interest in the subject should be given a realistic estimate of the cost of the medical services that are recommended. The initiative for bringing about this discussion rests with us, because many patients are hesitant to broach a topic which may be of utmost concern to them but which is secondary to the purpose of their visit. In the event that the ordinary cost of our proposed services would result in a hardship to our patient, we are obligated to provide him with adequate medical service at a fee that he can afford to pay.

For many years the application form for membership in this Society has contained a pledge under which the applicant signs his name. This pledge contains the following sentence: "I also agree not to participate in

fee-splitting or any other form of unethical medical practice." All but the oldest members of this Society have signed this pledge, and those who were not given this opportunity have over the years demonstrated their adherence to the principles which it embodies. Our opposition to any form of unethical practice is a matter of record and does not warrant extensive comment at this time.

A great deal of prominence, more perhaps than the subject merits, has been given by the lay press to the performance of unnecessary surgical procedures. Within the past year, during a panel discussion at a large medical meeting, one of this country's best known surgeons and teachers predicted that fewer hemorrhoidectomies would be done when unnecessary operations have been eliminated. This prediction may be correct, but one should not infer from these remarks that the proctologist is the one who is responsible for performing unwarranted surgical operations. He is no more to be blamed than can the gynecologist be held accountable for the needless hysterectomies for which the profession is currently criticized. It is the surgeon who has little or no interest in the diagnosis and treatment of proctologic disorders, and frequently the one who disparages proctology as a specialty, who is responsible for the unwarranted surgical procedures which are employed for the treatment of conditions which would readily respond to less radical therapy.

What constitutes good medical care, aside from its scientific aspects, has been the basis of the preceding remarks. Admittedly what has been said reflects a personal opinion and it is not to be expected that it will be met with complete agreement by every listener. However, the thoughts that have been expressed are based upon principles which will enable us to provide medical care that is contrived for the welfare of the patient rather than for the interests of the practitioner.

Since the final quarter of the last century when a few foresighted physicians became interested in a field of medicine exploited by irregular practitioners and neglected by those who were indifferent to its potentialities, proctology has become firmly established in the group of recognized medical specialties. A few physicians whose surgical interests appear to be broader than their concept of the type of medical care to which the patient is entitled even now maintain that there is no justification for a specialty as restricted as ours. Discredit is often reflected upon our specialty by those who refer to themselves as proctologists but who adhere to systems and methods of therapy inadequate for all the conditions that they profess to treat.

To refute any criticism that may be directed toward our specialty we, as members of this Society and as individual practitioners, must continue to provide the kind of medical service to which our patients are rightfully entitled.

It is not the public expression of our approval of lofty principles that is the true measure of our desire to meet the responsibilities that are ours. The only adequate test of our sincerity in seeking to discharge our obligations properly is the manner in which we serve those who turn to us for medical attention. The strong foundation upon which our specialty has been established is a tribute to those who have preceded us. It is incumbent upon us to persist in our efforts to provide the highest quality of medical care if we are to merit the gratitude of our patients and command the respect of our fellow practitioners.

W. W. Green, M.D.
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Advances in Surgery of the Colon

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FOR many years we have appreciated the value of proctology as a subspecialty of surgery. Because of this conviction we encouraged the establishment of a division of proctology in the Department of Surgery at Tulane University and in the Ochsner Clinic and are convinced that this was an extremely constructive move.

When the changes which have been made in the past ten years in the treatment of lesions of the large bowel are reviewed, they can be designated as being both more radical and yet more conservative. In any instance, real progress has been made and it is now possible to obtain results which were not possible before. Certainly this is necessary because otherwise progress would not be made. Whereas the improvement in results is likely to be credited to such innovations as antibiotics, and certainly they do deserve their share of credit in making possible today procedures which were not possible before they were available, one cannot give credit to any one modality; a number of factors must be credited for the advances made. It is for this reason that it is difficult at times to evaluate the relative merits of each. Although I am convinced that the better evaluation of the patient with a lesion of the intestinal tract and his better preparation for operation by correcting the blood volume and hemoglobin deficiencies, the availability of better anesthesia and better postoperative care have done much to improve the results in the treatment of lesions of the colon, I cannot agree with those who maintain that the use of antibiotics in the preparation of patients undergoing surgery of the intestinal tract is of no value. However, no antibiotic can replace good surgical technic; and even though antibiotics are used, it is imperative that good surgical principles be followed. I am equally convinced that procedures can be used safely following adequate preparation of the bowel with antibiotics which would have been associated with some hazard before antibiotics were available. I refer particularly to the almost routine use of open anastomoses at the present time. Before the availability of efficient intestinal antiseptics, stage operations were frequent and closed or aseptic anastomoses were the rule rather than an exception. It was the experience of individuals treating many patients with colonic lesions that only by utilization of these maneuvers could the best results be obtained. However, with the better preparation of the bowel by means of intestinal antiseptics, stage operations have been almost completely eliminated except in patients with obstruction and open anastomoses have become the rule. In the preparation of a patient for surgery of the intestinal tract it is important whenever possible to cleanse the bowel mechanically. Although there has been very little change in the method of doing this within the past decade, this offers little or no problem in the individual who has no obstruction or in whom there is little or no inflammation. Even in the patient with incomplete obstruction it is possible safely to cleanse the bowel mechanically by repeated administration of small amounts of a saline laxative. In such a patient it is our custom to give 1 gm. of magnesium sulfate every one to two hours until six doses have been given. In this way a liquid stool is obtained which will usually pass an incomplete obstructing lesion without any difficulty. Following mechanical cleansing of the bowel, the patient should be placed upon a non-residue diet of sufficient caloric intake to prevent loss of body tissue. This should be relatively high in protein and carbohydrate. Intestinal antiseptics should

be administered and these preferably should be absorbed not at all or relatively little from the intestinal tract since the action of the antiseptic is needed only in the bowel lumen. Whereas previously a large gamut of substances was used, it is now quite universal that a combination of one of the sulfonamides, particularly sulfathalidine® or sulfasuxidine,® be combined with neomycin.® The administration of 2 gm. of sulfathalidine four times a day for three to four days preoperatively, followed by the administration of 1 gm. of neomycin every four hours for the twenty-four hours preceding the operation, will effectively sterilize the bowel and not only rid it of coliform organisms but also, as emphasized by Poth,[1] prevent the outgrowth of Aerobacter aerogenes. Intestinal antiseptics must be used with caution, however, and should not be prolonged over long periods of time because of the danger of production of the so-called "antibiotic bowel" which results from the overgrowth of yeast and frequently staphylocci in the intestinal tract in which the normal flora have been eliminated. Since non-pathogenic bacteria in the colon are necessary to prevent the overgrowth of these organisms, there is probably less danger of the development of the so-called "antibiotic bowel" in the short preparation of patients for surgery of the intestinal tract than in patients in whom antibiotics are given over long periods of time for other conditions.

As desirable as aseptic anastomosis was before the availability of suitable and efficient intestinal antiseptics, it cannot be denied that the approximation of the two portions of the bowel could not be done as carefully as can the meticulous layer approximation which is possible with open anastomosis. It is well known that in order to obtain a minimal amount of scar, careful approximation of the various layers, particularly the epithelial layer, is imperative. When one desires a minimal cutaneous scar, the skin edges are meticulously approximated with fine sutures; and although a scar on the face may be undesirable, a scar on the mucous membrane of a hollow viscus such as the colon is as equally undesirable if not more so because considerable scarring with its cicatricial contraction can produce varying degrees of obstruction of the relatively small lumen of the hollow viscus. It is only by open anastomosis and careful layer approximation of the two portions of the bowel that a minimal scar can be obtained. In all intestinal anastomoses for the past five or six years we have employed the open technic using a two-layer approximation with cotton. For the seromuscular layer, a mattress suture of quilting cotton is used and a very fine cotton thread, No. 120, is used to approximate the mucous membrane. By the careful apposition of the mucous membrane a minimal scar is obtained and practically no narrowing of the lumen results.

Concerning the value of open anastomosis, it is interesting to recall a statement made in 1948 by Graham[2]: "Controversy continues concerning the relative merits of closed and open methods of intestinal anastomosis. Despite strenuous criticism by Whipple, Meyers, and others, of the aseptic technics, particularly those that employ special clamps, their popularity has grown steadily since 1937, when a survey made by us revealed almost universal preference for the open anastomosis. Stone, Rankin, Wangensteen, Waugh, White, Pemberton, and Dixon are among those who believe mortality is favorably influenced by the closed technique. Coller, who says that the aseptic method of anastomosis has become increasingly popular with their surgeons, believes that the striking point of difference between the two methods is not that peritonitis is more apt to occur with one or the other, but that with any open anastomosis there is a high risk of contamination of the abdominal wall which withstands infection less well than the peritoneum." I am sure that most, if not all, of the men quoted by Graham only six years ago employ open anastomoses at the present time more frequently than closed anastomoses.

Another advance which has been made within recent years is more radical extirpation of lesions of the left side of the colon. It is of interest that for some time the end results following surgical extirpation of right-sided lesions have been better than those following extirpation of left-sided lesions. It was previously stated that lesions on the left side of the colon grew more rapidly, metastasized earlier and because of this usually had a poorer prognosis than lesions of the right colon. There is considerable doubt in my mind whether the poorer prognosis following the treatment of lesions of the left colon has been due to difference in behavior of the lesion itself or rather to the fact that previously less extensive excisions were employed on the left side than on

the right. Whereas few right-sided lesions were treated except by resection of the terminal ileum, all of the right colon and the right portion of the transverse colon with the re-establishment of the continuity of the intestinal tract by an ileotransverse colostomy (Fig. 1), generally lesions on the left side of the colon were treated by segmental resection (Fig. 2), an operation which obviously cannot remove as much bowel and lymph-bearing area as does the more radical procedure of hemicolectomy (Figs. 3 and 4). Moynihan,[3] as early as 1908, advocated high ligation of the inferior mesenteric vessels for left-sided lesions and emphasized that in the removal of these lesions it was necessary always to remove the lymph node which he found present at the origin of the inferior mesenteric artery. In spite of these early teachings of Moynihan, it is only recently that the left hemicolectomy has been performed and the inferior mesenteric artery ligated at its origin at the aorta in the treatment of rectosigmoid lesions. Grinnell[4] advocated inferior mesenteric vessel ligation at the point of origin in abdominoperineal resections for carcinoma of the rectum and for resections done for lesions above the rectum. Toupet[5] advocated inferior mesenteric vessel ligation at the aorta and performed pull-through operations for low-lying lesions. He states that there is no danger of interference with the blood supply when this is done because actually the left colon is better vascularized through the mid-colic than through the inferior mesenteric and sigmoidal vessels. He is of the opinion that the higher the level of resection and the closer the resection is to the origin of the mid-colic artery, the more adequate the blood supply will be. Others who have advocated high ligation of the inferior mesenteric vessels are Deddish,[6] Ault et al.,[7] State,[8] Welch and Rheinlander,[9] and Grinnell and Hiatt.[10] Grinnell and Hiatt[10] found in 12 per cent of the tumors of the left colon and rectum and in 21 per cent of tumors of the descending colon there was evidence of the need for higher ligation of the vessels. In a recent study by McElwain, Bacon and Trimpe[11] it was shown that by careful examination of the lymph nodes that were resected in ninety cases of cancer of the colon in which the left colon was removed, twelve had involvement of the nodes near the origin of the inferior mesenteric artery without evidence of liver metastasis. In four of these the nodes possibly could have been extirpated by ligating the inferior mesenteric artery just distal to the left colic branch. The remaining eight, however, could not have been adequately removed except by high ligation. Thus positive nodes would have been left behind in 9 per cent of all operative cases if high ligation had not been performed. Grinnell[4] found involvement of the nodes at origin of the inferior mesenteric vessels in 17 per cent. It is quite obvious that better results can be and are being obtained in lesions of the left side of the descending colon, sigmoid and rectosigmoid by left hemicolectomy than by segmental resection.

Another popularization of an old technic is the performance of abdominoperineal resection using two teams working simultaneously, one working perineally and the other within the abdomen. Hill[12] states that in 1906 Bloodgood reported simultaneous operative dissection through abdominal and sacral incisions in an individual with carcinoma of the upper sigmoid and rectum with restoration of continuity. Kirschner[13] in 1934 called attention to this technic of abdominoperineal resection. Trueblood[14] in 1935 also described the method. Welch and Rheinlander[9] employ the two-team technic for the performance of the radical excision and pull-through technic. There are many who believe that the employment of the two-team technic is undesirable because, presumably, it is associated with a greater degree of trauma and is followed by a higher incidence of shock than when the abdominal and perineal procedures are separately performed. However, this has not been our experience. Because of the shorter time necessary to effect an abdominoperineal resection and because it is possible to do a more radical procedure using two teams, we prefer the two-team technic to the older method of performing either the abdominal portion or the perineal portion first to be followed by the other after completion of the first. The criticism that bleeding is greater we have not found to be true because of the greater facility with which bleeding can be controlled when the two teams are working simultaneously.

In order to prevent contamination of the bowel wound with tumor cells and thus decrease the incidence of local recurrence, Cole[15] and McGrew et al.[16] advocate excluding the tumor from the anastomotic site by ligating the bowel above and below the tumor before

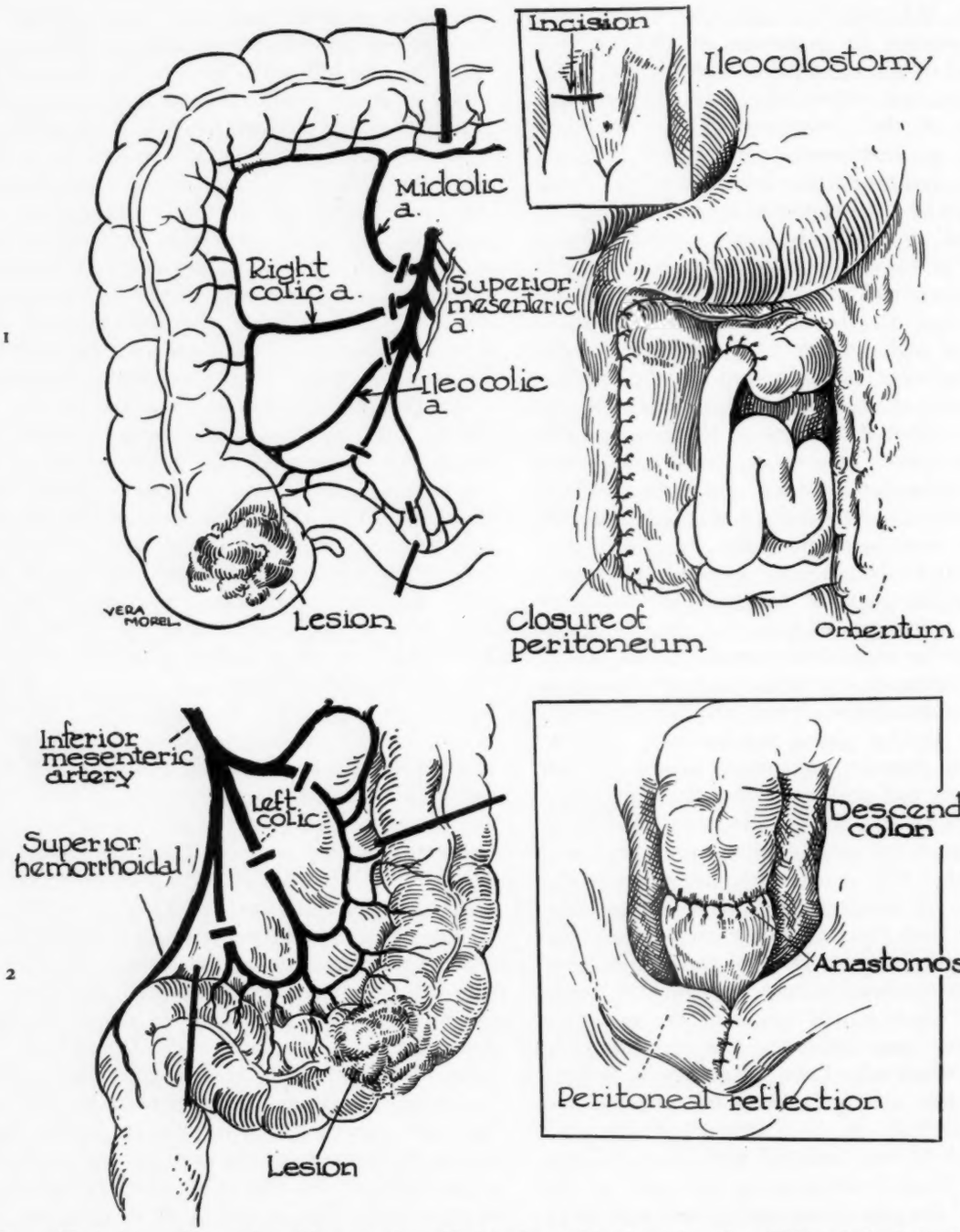

FIG. 1. Technic usually employed for resection of lesions located in the right colon. Although the lesion is limited to the cecum, radical resection of the terminal ileum, cecum, descending colon and right transverse colon is performed. In this way a considerable lymph node–bearing area is removed. Re-establishment of intestinal continuity is secured by end-to-end anastomosis between the terminal ileum and the transverse colon.

FIG. 2. Diagram showing previously used method of segmental resection for lesions of the left colon. Although in most instances most of the tumor-bearing portion of the bowel was removed, there was an incomplete resection of the lymph node–bearing area since in many instances lymph node involvement up to the origin of the inferior mesenteric vessels occurs.

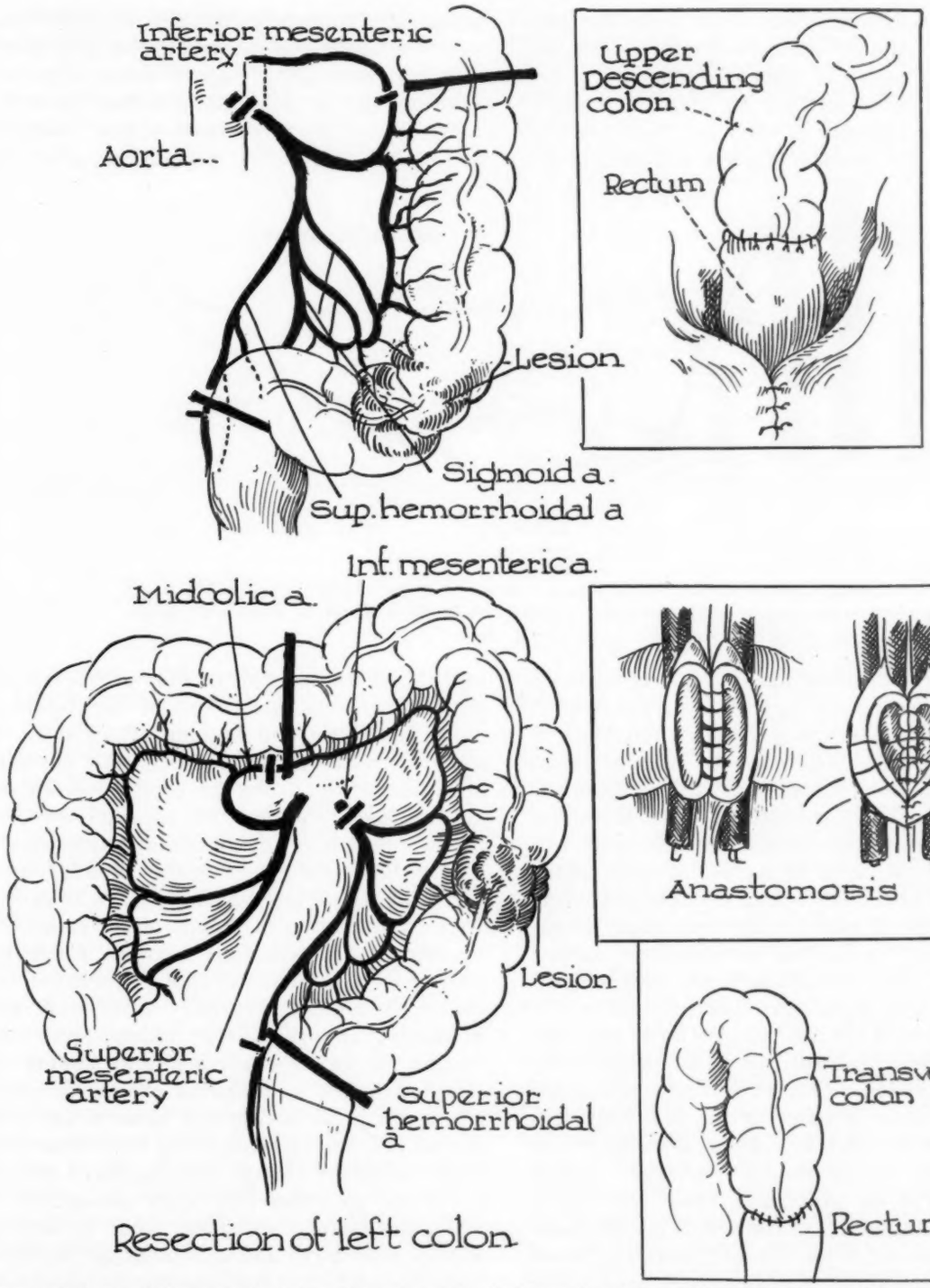

FIG. 3. Left hemicolectomy for lesion of the sigmoid, which permits wide excision of the involved bowel as well as wide excision of the lymph node–bearing area.

FIG. 4. Resection of sigmoid, descending colon, hepatic flexure and left transverse colon for lesions in the descending colon. Re-establishment of continuity by end-to-end anastomosis performed by two-layer technic. Careful approximation of the mucous membrane.

any manipulation is done. (Fig. 5.) In this way any desquamated malignant cells which are discharged into the lumen of the bowel are prevented from getting to the anastomotic site and the danger of their implantation and growth at the anastomotic site is diminished.

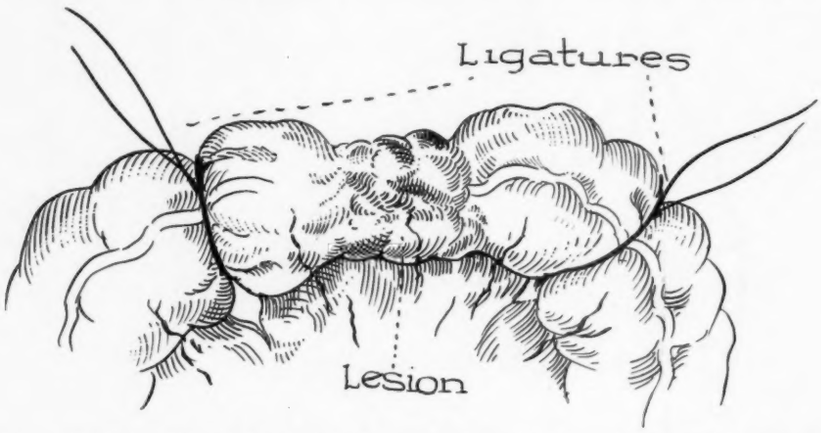

FIG. 5. Technic of Cole of isolating the tumor from the site of division of the bowel in order to prevent implantation of tumor cells which might be extruded into the lumen of the bowel into the anastomotic site.

In order to decrease the incidence of infection and subsequent scarring and cicatricial stenosis of a colostomy opening Crider[17] and Butler[18] advocate careful suture of the mucous membrane of the colon to the skin at the time of the performance of the colostomy. This method has recently been advocated by Lemmer and Mehnert.[19] In order to prevent stenosis in the ileum and to provide a long enough protruding loop to extend into an ileostomy bag, Dragstedt et al.[20] suggested covering the serosal surface of the protruding ileum with a split-thickness skin graft and reported good results from its use. This has become quite popular. This technic has recently been modified by Crile and Turnbull[21] in which they emphasized the importance of covering the ileal loop with epithelium in order to prevent serositis which can produce an "ileostomy syndrome." Unless the serosa of the protruding bowel is covered, severe inflammation of the serosa results causing marked inflammation of the entire bowel and the production of rather severe mesenteric lymphadenitis and associated ileus. As a result of the swelling, first edema and later scarring, obstruction of the ileum occurs and the patient becomes extremely ill. In order to prevent this, Crile and Turnbull[21] recommend excision of the serosa and musculature of the distal portion of the protruding ileum, following which the remaining mucous membrane is folded back over the bowel and sutured to the skin. In this way the protruding loop of ileum is covered with mucosa on its external surface as well as in the lumen. By this procedure the "ileostomy syndrome" has been completely eliminated and the necessary wait of four or five months for the mucous membrane of the bowel to cover the ileum and become attached to the skin is obviated, during which time considerable amount of fibrosis can result, and scarring and stenosis of the ileostomy are likely to occur.

The role which adenomatous polyps play in the production of cancer of the colon is becoming more appreciated as experience is gained. In a consideration of polyps of the colon it is necessary to differentiate between congenital polyposis and acquired polypi because the former is almost invariably associated with malignant change if the patient lives long enough or unless the colon and lesions are removed before a malignant change develops. There are probably several reasons why congenital polyposis is more likely to be associated with malignant change than acquired polypi: (1) They are inherently more susceptible to malignant change. (2) They occur at birth or shortly after birth and exist a longer period of time favoring the development of malignant change. (3) Because of the large number of them, there is a greater possibility of malignant change. As a matter of fact, it is not unusual for cancer to develop in many sites in such a bowel. At the present time there is some controversy whether total colectomy should be done in these cases with the production of

an ileostomy or whether one is justified in leaving the rectum and distal sigmoid and employing proctoscopic removal of the polypi in the remaining segment. Whereas ten years ago most surgeons advocated total colectomy in all cases, at the present time there is a trend

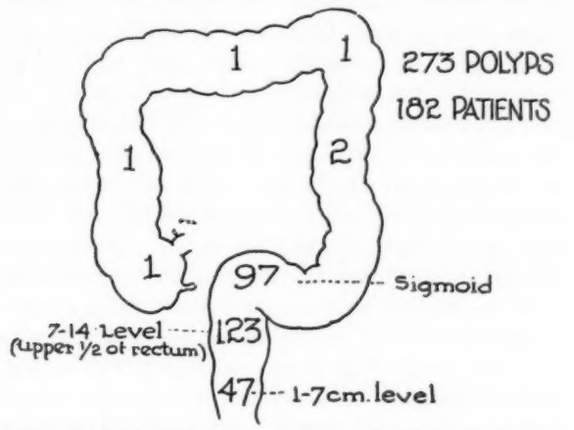

FIG. 6. Incidence of occurrence of polyps in 2,000 routine proctoscopies; 273 polyps were found in 182 patients.

toward greater conservatism; namely, removal of all the colon down to the distal sigmoid with the performance of an ileosigmoidostomy, following which the polypi in the distal colonic segment are removed. This permits frequent examinations of the distal colonic segment and removal of any polypi which subsequently may develop. If, however, malignant lesions do develop in the rectosigmoidal segment, which is likely, radical removal of this segment is obligatory.

Of the acquired polypi, it is necessary to distinguish between those which occur spontaneously and those which follow inflammatory disease such as ulcerative colitis. Of these two, polypi which occur spontaneously are more likely to undergo malignant change than those which follow an inflammatory process. There are two types of polypi which occur spontaneously, the adenomatous polyp and papillary adenoma or villous tumor. Of the two, the adenomatous polyp is more frequent, villous tumors or papillary adenomas being relatively infrequent. Acquired papillomas are of extreme importance because of their frequency, the incidence varying in routine proctoscopy from 17 per cent reported by Enquist and State,[22] 9.5 per cent reported by Helwig[23] in 1,460 autopsies, to 6 per cent reported by Berkheimer.[24] Enquist and State[22] found in a total of 3,364 patients examined that 580 (17.2 per cent) had rectal or colonic polyps; 576 were discovered by proctoscopy and four by barium enema. Between the ages of forty-five and sixty-four, 18 per cent of all patients examined had polypi, whereas these tumors were found in

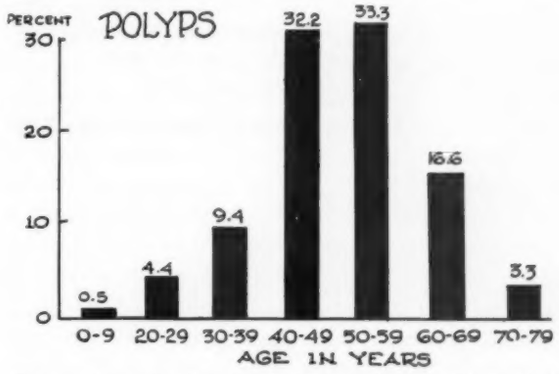

FIG. 7. Age distribution in 182 patients with benign polyps detected on routine examination.

only 12 per cent of those over sixty-five. They emphasized the importance of repeated examinations at annual visits because in about 12 per cent of patients new tumors develop within a year. Swinton[25] states that they have found polyps in 25 per cent of all the surgical specimens removed for cancer of the colon at the Lahey Clinic. Among 470 cases of polyps of the colon and rectum, 436 were adenomas and 34 were papillary adenomas or villous tumors. At the New England Deaconess Hospital from 1935 to 1945, in a series of 1,843 consecutive autopsies benign mucosal polyps of the rectum and colon were found in 7 per cent of the cases, of which 58 per cent were single and 42 per cent were multiple. They found 5 per cent among all patients over thirty-five years of age undergoing sigmoidoscopy. On our Proctologic Service at the Ochsner Clinic, in 2,000 routine proctoscopies 273 polyps were found in 182 patients (9.1 per cent). (Fig. 6.) Thirty patients had multiple polyps. Of the 182 patients with polypi, the age distribution was as follows: ten to nineteen, 0.5 per cent; twenty to twenty-nine, 4.4 per cent; thirty to thirty-nine, 9.4 per cent; forty to forty-nine, 32.2 per cent; fifty to fifty-nine, 33.3 per cent; sixty to sixty-nine, 16.6 per cent; and seventy to seventy-nine, 3.3 per cent. (Fig. 7.) Thus two-thirds of the polypi found on routine proctoscopy were in the fifth and sixth decades of life. Since polypi are definitely premalignant lesions, it is obvious that routine

proctoscopic and sigmoidoscopic examinations should be done in all persons between the ages of forty and sixty years. In the same 2,000 routine proctoscopies, forty-two (2.1 per cent) malignant tumors were discovered. (Fig. 8.)

The treatment of polyps consists of surgical

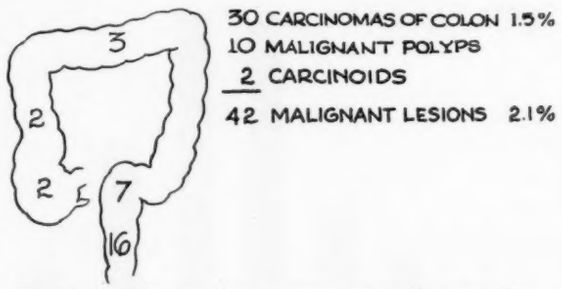

FIG. 8. Incidence of occurrence of malignant disease in 2,000 patients examined routinely.

extirpation because they are distinctly pre-malignant. Lahey and Swinton[26] state that all polyps must be considered as malignant lesions. Swinton and Warren[27] in 1939 reported that at least 14 per cent of 827 cases of carcinoma of the colon arise in pre-existing adenomas, and Helwig[23] believes that the majority of carcinomas arise in pre-existing adenomas. Sandusky and Parsons[28] report that 8.9 per cent of 202 cases of adenomatous polyps showed definite malignant change. Of 108 solitary polypi, 9.2 per cent had biopsy only, 87.9 per cent local excision and 2.8 per cent resection. Of forty multiple polypi, 12.5 per cent had biopsy only, 80 per cent had local excision and 7.5 per cent had resection. Of six diffuse polypi, 50 per cent had biopsy only, none had local excision and 50 per cent had resection. In the 202 patients, 156 (77.2 per cent) had benign polyps. There were twenty-eight (13.8 per cent) in which there was a benign polyp plus a large bowel cancer, and there were eighteen (8.9 per cent) in which a polyp showed malignant change. In the distally located lesions in which there is carcinoma *in situ* or in which there is non-invasive carcinoma, one is probably justified in doing a radical local excision which consists of removal of the polyp and extensive cauterization of the base. In all other polypi, such as those that are invasive in the distally located lesions, abdominoperineal resection or wide local excision should be done.

Papillary adenomas are a peculiar type; they are particularly likely to recur but are less likely to be invasive. In a series of patients reported by Sunderland and Binkley[29] 68.7 per cent showed cytologic evidence of carcinoma either in the original tumor or in recurrence at the site of removal; 39.5 per cent developed invasive carcinoma. Welch, McKittrick and Behringer[30] found that of twenty-seven cases of papillary adenoma, six (22 per cent) had cancer. Of sixty-six sessile adenomatous polyps, fourteen (21 per cent) had cancer. Of 229 pedunculated or indeterminate, forty (17 per cent) had cancer. Of the total 322, sixty (18 per cent) had cancer. Of 222 polyps in the rectum, thirty-six (16.6 per cent) had cancer; of 102 in the colon, twenty-four (23.6 per cent) had cancer. In 1,460 consecutive autopsies reported by Helwig[23] the incidence of adenomas in Negro patients was only 2.7 per cent, whereas in 1,279 white patients it was 10.4 per cent. The peak incidence was in the eighth decade in which 25.8 per cent were found in men and 20.5 in women. In all decades above the third men were slightly more susceptible to these lesions than women. The incidence of malignant change in Helwig's[23] cases was as follows: In the entire series of 1,460 cases, 139 adenomas were found of which 10 (11.2 per cent) showed evidence of malignant change in the adenoma; in eight of the ten the malignant change occurred in only one adenoma, whereas in two it was present in two and three adenomas, respectively.

In addition to adenomas which occur spontaneously there are the polypoid lesions which are associated with and follow inflammatory disease of the colon. Helwig,[23] in a series of 1,460 consecutive autopsies in which the colon was carefully studied for adenomas, found 139 (9.5 per cent); however, in only one instance was there an association of colitis and adenoma. Helwig states, "It would appear from this series that any relation between the presence of some form of colitis and adenomas is purely coincidental." Although Helwig[23] questions the fact that adenomas have any association with inflammatory disease, Rice and Trueblood[31] found among 129 cases of ulcerative colitis (which incidentally was the largest series reported in England at that time) that four (3 per cent) developed carcinoma at an average age of thirty-seven as compared with an average age of fifty-three in which carcinoma not associated with ulcerative colitis developed. Johnson and Orr[32] reported two cases of carcinoma developing in patients with chronic

ulcerative colitis, one thirty-two years old and the other forty-three. The incidence of malignant change in ulcerative colitis in the Cleveland Clinic was 3.8 per cent as reported by Gleckler and Brown.[33] The average age at onset was 44.3 years. The incidence reported by Lyons and Garlock[34] was 3.9 per cent. They found that of all patients having ulcerative colitis for twelve years, carcinoma developed in 36 per cent, whereas in sixteen cases in which the ulcerative colitis involved the *rectum* for over twelve years, carcinoma developed in seven or 43 per cent. Lahey[35] believes that chronic ulcerative colitis predisposes to malignant change, but states that whether one accepts this or not, the fact remains that the prognosis is bad when carcinoma is found in individuals with ulcerative colitis. Of eighteen patients in his series in which ulcerative colitis and carcinoma occurred concomitantly, only two were alive without recurrence and these two could hardly be classified as clinical carcinomas because the carcinoma was found only on microscopic examination. Shands, Dockerty, and Bargen[36] found that the average age at which adenocarcinomas developed in patients with ulcerative colitis was forty-two and the average duration of the inflammatory lesion before carcinoma developed was 15.3 years. In their series 11 per cent had ulcerative colitis less than five years before the carcinoma was discovered and 24.7 per cent had it less than ten years. They agree with Lahey[35] that the type of carcinoma associated with ulcerative colitis is a highly malignant one. Thirty-nine of eighty-eight separate adenocarcinomas were graded as 3 or 4 (Broders) and twenty-five of seventy-three patients had mucous adenocarcinomas. Of the entire group in which there was associated carcinoma and ulcerative colitis there were only two five-year survivors, and subsequently one of these died from a second adenocarcinoma of the colon. Fifty-seven of the seventy-three were known to have died of carcinoma of the large bowel and fifteen had been followed up less than five years. Shands et al.[36] also emphasize the importance of multicentricity in these cases. In forty resected specimens there was evidence of multicentric neoplastic lesions in twenty-one, and in this group there was a high incidence of pseudopolyposis (sixteen of the twenty-one, or 76.2 per cent) as contrasted with only 15 per cent in which the lesions were not multicentric. Because of the high mortality rate, the authors believe that colectomy should be done even though the lesion may remain quiescent for ten years or longer. Counsell and Dukes[37] found that the incidence of carcinoma in patients with ulcerative colitis treated surgically was 11.1 per cent. In discussing the controversy among authorities as to whether there is an association between ulcerative colitis and the incidence of cancer, they state that the confusion is due to the fact that many investigators fail to distinguish between the different types of lesions occurring in chronic ulcerative colitis; that is, mucosal tags, inflammatory polyps and adenomatous polyps. The most common varieties in ulcerative colitis are the mucosal tags and the inflammatory polyps which they believe are not precursors of cancer. On the other hand, adenomatous polyps are definitely precancerous. Their series also shows the relationship of the duration of the disease to the development of cancer. Although there were seven cases of carcinoma among sixty-three of the entire group (11.1 per cent), in those with a history of less than ten years there were fifty-two cases, of which only two had carcinoma. On the other hand, there were eleven with a history of ulcerative colitis for more than ten years, of which five had carcinoma. Thus carcinoma developed in almost half of those with ulcerative colitis for more than ten years. Bargen, Sauer, Sloan and Gage[38] showed that there is a definite relationship between the development of cancer and chronic ulcerative colitis. In a previous investigation in 1950 in which a series of 2,000 cases of chronic ulcerative colitis at the Mayo Clinic were studied, polyposis of the colon was present in 19.4 per cent. A study was made of the follow-up of these cases with the exception of those who were fifty years of age or older at the time they were admitted to the Clinic or those who died in the hospital after the admission. There were 1,564 patients who were less than fifty years of age when first admitted to the Clinic and who have been traced for one or more years and were free from malignant disease for at least a year. In this group ninety-eight cases (.06 per cent) developed cancer of the rectum or colon and died. Bargen et al.[38] conclude that the younger the patient, the greater the likelihood of developing carcinoma; they believe that cancer occurs

thirty times more frequently in patients with ulcerative colitis than in those of the general population of a similar age. Jackman and Mayo[39] state that polyposis occurs in from 10 to 12 per cent of patients with chronic ulcerative colitis and that from 2 to 3 per cent of the patients with chronic ulcerative colitis develop carcinoma of the colon.

Although a decade ago ulcerative colitis was treated conservatively as a rule by non-operative measures or conservative operative procedures such as ileostomy, at the present time colonic resection is being performed much more frequently in order to prevent the complications which follow ulcerative colitis. Cave's[40] studies demonstrate that results following ileostomy alone are not good. Of 101 patients operated upon over a period of ten years at the Roosevelt Hospital, there were twenty early or immediate fatalities. Of the eighty-one survivors, fifty had been recently examined. These had gotten along well in that there was a gain in weight but many of them continued to have symptoms. Because ileostomy and other conservative surgical procedures have failed so frequently, it is the consensus of most physicians at present that in the advanced fulminating case of ulcerative colitis surgical extirpation of the colon is a life-saving procedure. Whereas previously the sick patient with ulcerative colitis usually was considered too ill to have resection of the diseased bowel, at present such an individual is considered too sick *not* to have it because unless the highly inflamed, suppurative bowel is removed the patient's chances of recovery are not good. Ferguson and Erb[41] report seven cases in which resections were done, and Devine and Devine[42] reported eleven cases. In the former series all patients were extremely ill; six survived subtotal or near total colectomy, one a near colectomy combined with excision of the rectum, one a partial colectomy involving two-thirds of the colon, and one a first stage colectomy. The six were apparently cured. Dennis et al.[43] advocated vagus resection in the treatment of ulcerative colitis. In a series of twenty-two cases treated with vagus resection one died of cardiac complications without completion of the vagotomy, two were definitely worse, three were unchanged, fourteen seemed improved and three had not been operated upon long enough for evaluation. Eddy,[44] in a subsequent follow-up study of forty-two cases, concluded after a minimum three-year follow-up that vagus resection in ulcerative colitis is of value, but that patients with the disease for five years or more respond less favorably than those with disease of shorter duration. The proponents of vagus resection believe that its value consists of decreasing intestinal motility and spasm but they emphasize that although the patient may be symptomatically relieved, the disease may progress. However, Eddy warns that because of the danger of cancer developing in chronic ulcerative colitis, vagus resection should be employed cautiously.

McKittrick and Moore[45] emphasize that great individualization in patients with ulcerative colitis must be used and that once an ileostomy is done it can seldom be closed. They list the indications for colectomy as follows: (1) polyposis, (2) life-saving, (3) unsatisfactory response to ileostomy, (4) reactivation of the disease in the colon, and (5) a miscellaneous group in which other diseases are associated. Of their 149 patients with ulcerative colitis which had ileostomy as an initial or only procedure, twenty-two (14.8 per cent) died after operation. Most of these were due to progress of the disease (the question arises whether or not they might have been saved by colectomy). Eighty-seven (68.5 per cent) of those who survived had subtotal colectomy at intervals of a few months to twelve years after the ileostomy with a mortality of 6.9 per cent. Thirty-three (40.7 per cent) of those who survived the colectomy had removal of the rectal stump by combined abdominoperineal resection with a mortality rate of 3.3 per cent. Only five had a re-establishment of the intestinal continuity. Three of these did not respond favorably, two had a second ileostomy and are now in excellent condition, and one nearly died of his colitis and had remained a chronic invalid but recently died of a brain tumor. There were forty-five deaths (12 per cent) in the 372 cases. In those cases in which no operation was performed there were twelve deaths (5 per cent); ileostomy, 143 cases, twenty-five deaths (17.4 per cent); subtotal colectomy, eighty-seven cases, seven deaths (8 per cent); proctectomy, thirty-three cases, one death (3.3 per cent); restitution of continuity, five cases, no deaths. Follow-up on 110 cases showed that 84 per cent of the patients consider themselves in good health.

One of the conditions in which there has been considerable change concerning therapeutic management in recent years is diverticulitis of the colon. Previously most patients with diverticulitis were treated conservatively unless there was a surgical complication such as perforation and abscess formation or the production of a fistula or stenosis. It is probably understandable that before the age of antibiotics ultraconservatism was resorted to because of the greater hazard of extirpation of the bowel in such cases. Another change in concept concerning diverticulitis is that it is now appreciated that diverticulitis is frequently a cause of massive bleeding, whereas previously in such cases diverticulitis was seldom considered a cause. Diverticulitis is indeed a hazard, not only because of the possibility that it may progress, lead to perforation, fistula formation, cicatricial stenosis and severe hemorrhage, but also because it may be confused with carcinoma. Contrary to what was previously thought, there are probably few if any instances of malignant change in a bowel on the basis of diverticulitis. On the other hand, diverticulitis and carcinoma of the colon can occur concomitantly; and if diverticula are demonstrated, all too frequently the symptoms are considered as being due to the diverticulitis and the possibility of a neoplasm is disregarded.

Whereas previously ultraconservative procedures were used such as drainage of an abscess or proximal colostomy, it is now generally accepted that individuals with persistent symptoms of diverticulitis are best treated by resecting the involved bowel. Whereas proximal colostomy is still frequently necessary as a preliminary procedure in those cases in which there is considerable inflammation or obstruction, occasionally it is safe to perform primary resection of the involved colonic segment with immediate re-establishment of bowel continuity, either with or without complementary cecostomy. In cases in which a preliminary proximal transverse colostomy is necessary, several months should elapse before definitive resection of the involved segment is done if one can be reasonably sure that carcinoma does not coexist. In the period between the colostomy and resection, antibiotic therapy such as instillation of sulfathalidine daily into the distal colostomy opening and daily cleansing of the distal bowel should be done. Although the entire bowel may be involved in the diverticular process, resection of the whole colon is seldom necessary. Generally wide resection of the involved segment such as the sigmoid flexure or left hemicolectomy will effect a cure. The reason why incomplete removal of all the colon containing diverticula is successful is that the inflammatory segment is removed, and by removal of a considerable portion of the colon constipation is relieved and there is much less chance of the remaining diverticula becoming involved because stasis and inspissation of feces, which are the precipitating causes of diverticulitis, are prevented. In 1942 Smithwick[46] found in a review of the literature that of 1,332 reported cases of diverticulitis 28.5 per cent had some type of operative procedure. In his own series at the Massachusetts General Hospital 19.2 per cent had operation. He further found that operative procedures that did not divert the fecal stream or remove the involved bowel segment produced unsatisfactory results in 40 per cent. The fact that 12 per cent of Smithwick's patients submitted to resection of the involved segment had unsatisfactory results is of little significance because in many the procedure was done in patients with advanced disease. However, the late mortality rate was lower than in cases treated by other methods. In addition to Smithwick, Boyden,[47] Lloyd-Davies,[48] Ford,[49] Bartlett and McDermott,[50] Lewis and Hurwitz,[51] and Ransom[52] have advocated extirpation of the involved segment of bowel in diverticulitis.

A relatively rare condition, the treatment of which has been materially changed within the past decade, is congenital megacolon. Prior to the contribution by Swenson[53] the most satisfactory form of therapy was sympathectomy. Many of these patients were relieved but the results in most instances were far from good. Although it had long been recognized that there was a partial functional obstruction at the rectosigmoidal junction, it remained for Swenson to outline the rational therapy, which consists of resection of the narrowed rectosigmoid and varying lengths of the sigmoid and descending colon. Excellent results are being obtained by this technic. Excellent work on this subject also has been done by Hiatt.[54] It apparently makes little difference whether bowel continuity is obtained by suture within the abdomen or whether a pull-through procedure is used.

Another condition which is considered extremely rare but which we believe occurs more frequently than commonly thought to be is carcinoid of the rectum. We have had eighteen proven cases of rectal carcinoid within the past five years and it is of interest that the incidence is apparently increasing; this indicates that these lesions were previously missed or misdiagnosed. In our proctologic division more cases have been seen in the last two years than at any time previously. Many of the lesions are small and can be treated satisfactorily by radical removal through the proctoscope. In large lesions, however, it is necessary to perform an abdominoperineal resection.

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The Treatment of Malnutrition with Particular Reference to the Surgical Patient

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MALNUTRITION and outright starvation are still common clinical problems, especially in general surgery. In extreme instances the limitations imposed by the patients' nutritional status are usually evident but in other situations subclinical or rapidly developing deficient states may be difficult to recognize. Furthermore even with the clinical condition defined there often is confusion regarding the significance of nutritional deficiency in relation to the patient's convalescence and what to do about improving the situation. The clinician's difficulty arises from the complexity and variability of the individual problems; the failure of nutritionists and biologists to define clearly the relation between protein metabolism, caloric intake, the availability of body fat stores, hormonal influences and particularly limitations in the ability to meet caloric needs parenterally where this is important.

Whenever the gastrointestinal tract is in working order more can be accomplished by utilizing this route than by attempting to feed parenterally. For example, in preparing a patient with carcinoma of the esophagus for surgery it is better to utilize a nasogastric tube than to embark on a long-term parenteral feeding program that is cumbersome, laborious and probably less beneficial. The simplest approach is usually the best. Having established an artificial feeding device, the problem of what to put into the stomach or intestine arises. Once again the simplest measures are the most effective. There is seldom a patient that cannot be fed with a blenderized meal such as appears on the regular hospital tray. It is cheap and more satisfactory than any artificial formula we have tried. This feeding mixture can be supplemented with vitamins and minerals. The fluid volume can be adjusted to suit the patient's needs. We find intermittent small feedings more satisfactory than either continuous drip or large meals.

When the gastrointestinal tract is not available for nourishing the patient, one must turn to the intravenous route. What needs to be given depends upon the condition of the patient. It is common experience that in a previously well nourished individual short-term restriction of protein and caloric intakes is well tolerated. Ketosis can be prevented by as little as 50 gm. of dextrose daily. This amount of glucose will also reduce protein breakdown by as much as 50 per cent under some circumstances.[1] Under such conditions it is meddlesome to administer more than that amount of fluid necessary to cover the patient's fluid, electrolyte and minimal carbohydrate needs.

The situation is more complex when a long period of dietary restriction precedes an acute illness or when chronic states of limited or absent oral intake exist for weeks or months. Under these circumstances success or failure of a therapeutic program may depend upon our ability to meet nutritional needs that will allow reversal of the catabolic phase of illness.

The requirements for protein, calories, minerals and vitamins obviously vary depending upon individual circumstances. In general, about 2,000 calories and 50 gm. of protein are required to maintain a patient. Higher intakes of both protein and calories must be attained before significant protein synthesis can be expected. A caloric intake of about 1,000 calories per day supplied from dextrose and protein hydrolysates will maintain nitrogen equilibrium in the previously well nourished individual with ample endogenous fat stores and whose caloric restriction is of short duration.[2,3] On the other hand, in a depleted patient nitrogen equilib-

rium cannot be maintained on a 1,000 caloric intake regardless of the amount of protein supplied. Failure to take this fact into account has led to a generally pessimistic attitude on the part of clinicians concerning the possibilities of long-term maintenance of nutrition by the intravenous route. It is our opinion that the use of protein hydrolysates has not found its proper place in clinical practice because of the disadvantageous manner in which it is usually used.

Perhaps it is worth while to survey the means available to the clinician for supplying nourishment parenterally. Five per cent dextrose solutions are isotonic, non-irritating to veins, seldom pyrogenic, miscible with water soluble vitamins and electrolytes. They provide 50 gm. of sugar (200 calories) for each liter infused and 100 to 200 gm. of dextrose can usually be supplied along with a patient's daily fluid requirement. This quantity of sugar will prevent severe ketosis, lessen protein wastage and seldom be spilled out through the kidneys in appreciable amounts. Only rarely, however, can more than 600 calories be provided with this type of preparation, the quantity being limited by the fluid tolerance of the patient. Ordinarily nothing more is needed for short-term therapy. The caloric deficit is made up from fat and protein mobilized from tissue reserves.

The carbohydrate intake can be doubled by using 10 per cent dextrose in water solutions. These are hypertonic, irritating to veins and unless administered slowly, result in wastage via the urine. Assuming a fluid intake of 3,000 cc. daily, about 1,200 calories could be supplied from this source. This level of intake is respectable and is often sufficient, provided that some body protein and fat can be sacrificed without detriment.

The irritation to vein walls caused by hypertonic solutions sometimes becomes considerably important in a long-term parenteral nutrition program. The advantages of using solutions that may lead to thrombosis must be balanced against the possibility that all superficial veins will become sclerosed. Should this occur one may be faced with a major obstacle to the continuation of parenteral feeding. The use of plastic catheters threaded into larger vessels circumvents these difficulties temporarily but even then thrombosis is sometimes initiated by the use of irritating solutions. Thrombi in major veins may create additional hazards of embolism. Sepsis about inlying intravenous catheters is another problem that sometimes arises and when present creates a potential life-endangering situation. Intravenous catheterization may be an important aid in continuing a long-term parenteral nutrition program but should never be used for convenience of either the patient or the physician when peripheral veins are available.

The place of fructose and invert sugar in parenteral feeding is yet to be established.

Protein solutions are also available for parenteral administration. They are prepared by the enzymatic or acid hydrolysis of whole protein and are made up of individual amino acids and small aggregates of amino acids. They are usually available in concentrations of 5 to 10 per cent. Their content of utilizable protein varies from 70 to 100 per cent of the total amount present. These solutions can be administered in quantities up to 2 L. per day and concentrations up to 10 per cent. The higher concentrations cause occasional untoward reactions but these can be eliminated by slower rate of administration. By this means 50 to 200 gm. of protein can be supplied daily. Without entering into the question of nitrogen and protein metabolism immediately after operation or trauma, it can be stated in general that if sufficient calories to cover energy requirements are concomitantly supplied from other sources, parenterally administered protein can be used to rebuild tissue. If adequate calories are not supplied from other sources, protein supplied will be sacrificed to meet energy demands.

Alcohol has been employed for its caloric value. When completely metabolized it has a caloric value of approximately 7 calories per gram which on a volume basis amounts to 5.6 calories per cubic centimeter. The use of alcohol is limited by its action as a central nervous depressant. Alcohol can be administered in concentrations up to 10 per cent and an individual can metabolize about 10 cc. per hour. It is necessary to exercise rather careful control over the rate of administration in order to avoid toxic symptoms. Alcohol is used in conjunction with glucose or protein solutions and when used in this manner the supplementary calories appear capable of sustaining a positive nitrogen balance.[4] However, the limitations imposed by its toxic effects and the slow rate of

utilization make alcohol useful mainly as a supplement to other parenteral preparations.

In summary, the use of protein and carbohydrate solutions have been rather thoroughly explored both clinically and in the laboratory. Three inherent properties of these types of food impose sharp limitations upon their contribution to the caloric intake by the intravenous route. First, both protein and carbohydrate yield only 4 calories for each gram oxidized. Second, the osmotic effect of these types of molecules makes it impractical to raise their concentrations to higher levels because of irritation and thrombosis of veins that they produce. Third, they are readily excreted in the urine depending in large part on the concentration infused and the rate of infusion.

The most promising approach toward overcoming these difficulties is the development of fat emulsions suitable for intravenous administration. The chief advantage of the use of fat in parenteral nutrition resides in its contribution to the energy intake. Oil-in-water emulsions do not exert any osmotic effect and high concentrations can therefore be used without damage to veins. The fat is not excreted in the urine. These facts, coupled with the high caloric content of fat, 9 calories per gram, makes it possible to introduce considerable quantities of energy into relatively small volumes of fluid.

Ten to 15 per cent fat emulsions have had fairly extensive clinical trials. These preparations supply approximately 1,100 to 1,600 calories per liter. They have usually been administered in 500 to 1,000 mm. quantities daily. The fat passes from the blood into the liver and spleen principally where it is rapidly metabolized.[5–7] Properly prepared emulsions are not deposited intravascularly.

The immediate availability of intravenously administered fat for caloric purposes has been shown in several ways. Puppies have been reared with emulsified fat as their principal source of calories.[8] Animals and men have been maintained in positive nitrogen balance when the main source of energy was from emulsified fat.[9] Isotopically labeled fat administered in emulsions is quickly oxidized and a high percentage of injected radioactivity can be recovered in the expired carbon dioxide within a few hours.[10,11]

The use of fat emulsions to supply adequate caloric intake parenterally has permitted a limited examination of the practicability of complete long-term intravenous feeding. The following case report is presented to illustrate that an adequate supply of protein, carbohydrate fat, minerals and vitamins can be supplied and efficiently utilized to maintain nitrogen equilibrium.

CASE REPORT

M. M. (M.G.H. U No. 768575) was a fifty-six year old woman with chronic heart disease. An embolus to her superior mesenteric artery necessitated the resection of her entire small bowel distal to the second portion of the duodenum and right half of her colon. A duodenocolostomy was performed. Her immediate convalescence proceeded uneventfully, but she was never able to retain food or liquid in her stomach. Vomiting and watery diarrhea continued steadily even when nothing was taken by mouth. She lost weight rapidly. Approximately four weeks after operation a daily intravenous feeding program of 1,000 cc. of 10 per cent dextrose and water, 1,000 cc. of 10 per cent fat emulsion, 1,000 cc. of 10 per cent amigen®* and 500 cc. of 5 per cent dextrose and saline was begun. In addition she received 40 mEq. of potassium chloride and generous vitamin supplements in each daily ration. After one week of this daily feeding program the balance study summarized in Figure 1 was carried out. This was continued for eight days, and terminated when the patient had an aortic embolus. An embolectomy was performed but the patient died a week later of infection originating from a localized right gutter abscess which had been present since her original operation.

This case illustrates that a practical program for complete long-term parenteral feeding can be established. The essential features of this regimen were a daily intake of 100 gm. of protein and a caloric intake of 2,000 calories (including protein). The long period of depletion preceding this brief study, coupled with operation, infection, diarrhea and vomiting, qualify this patient as one of the most severe nutritional problems that will be faced. The 1,600 non-protein calorie level is near the basal requirement, and the 12 gm. daily nitrogen intake is low for optimal retention. This probably accounts for the fact that although equilibrium

* Amigen was supplied through the courtesy of Dr. Warren Cox, Mead Johnson Company, Evansville, Ind.

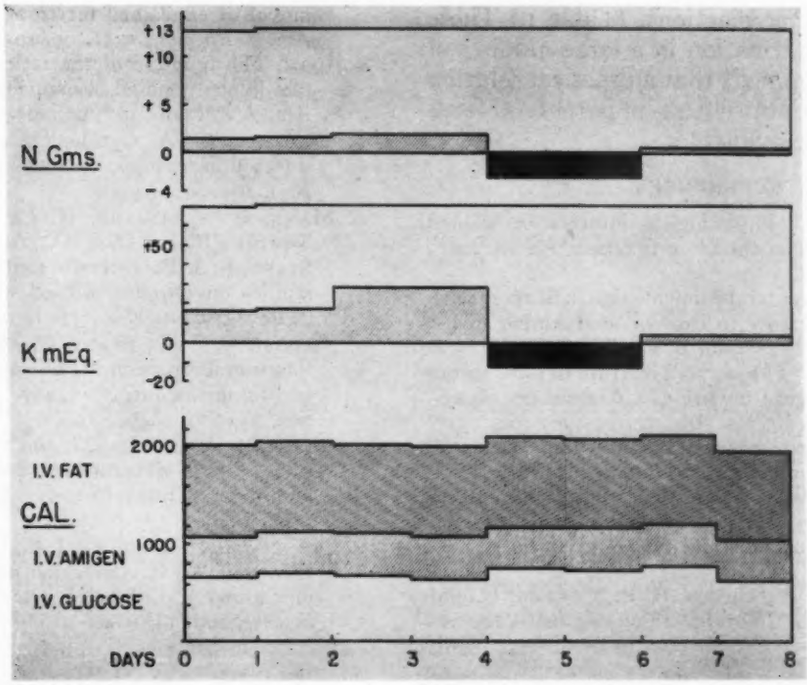

FIG. 1. Nitrogen and potassium balance during sixth week of total parenteral feeding. The intakes are charted upward from the zero line and the outputs are charted downward from the intake line. The two-day period of negative balance is unexplained. The calories from protein hydrolysates are included in the calculation of caloric intake at the bottom of the chart. This and other similar case studies will be presented in detail elsewhere.

was established, no significant nitrogen retention occurred. The contribution of intravenously administered fat to the caloric intake cannot be denied. Without this supplement the daily intake would have been approximately 1,000 calories, a level that precludes nitrogen equilibrium under the circumstances of this experiment.

Fat emulsions are not available at the present time because of technical difficulties in their preparation on a large scale and the occurrence of untoward reactions associated with their administration. In our hands and with our emulsions pyrogen reactions have occurred during 15 per cent of the infusions during the past three years and there has been a small incidence of other reactions. Recently technical advances in the preparation of emulsions have allowed the administration of 136 infusions to seventy-seven patients with a low incidence of

TABLE 1
REACTIONS TO INFUSION OF EMULSIFIED FAT*

Emulsions	Temperature Elevation					Chills	Back Pain	Number Infusions	Number Patients
	0–1°F.	1.1°–2.0°F.	2.1°–3.0°F.	3.1°–4.0°F.	4.1°F.+				
Cottonseed oil 4, batches....	92.6	7.4	0	0	0	2.2	2.9	136	77
All others, 116 batches.....	83.2	7.0	5.3	3.0	1.5	6.7	2.0	1,139	218

* Comparison of most recent emulsions used clinically (cottonseed oil) and all other emulsions used up until November, 1953. The figures are percentages of the total number of infusions.

pyrogen and other reactions. (Table 1.) These results need confirmation in a large number of patients but it appears that a practical solution to one of the major problems of parenteral feeding may soon be available.

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Hyaluronidase with Local Anesthesia in Anorectal Surgery

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FOR surgery of the anus and lower rectum local anesthesia by perianal infiltration is probably the safest.[1] This technic should be of advantage in proctologic surgery and is likely to prove especially useful for older and debilitated patients.[1]

The principal deterrents to the use of regional nerve block or local infiltration in operations on the lower rectum and anus have been (1) inadequacy of anesthesia, (2) insufficient duration of anesthesia, (3) distortion of the tissues at the site of the injection, (4) exquisite pain on injection until the anesthetic takes effect and (5) increased risk of infection.

In regional nerve block inadequate analgesia is usually the result of failure to deposit the anesthetic solution accurately and consistently along the nerve trunks. Even the most skilful anesthetist finds it impossible to produce an efficient block in every case.

Therefore, it appears that a method to facilitate diffusion of the anesthetic solution in the subcutaneous tissues will not only increase the efficiency of the local analgesia but may obviate the other undesirable factors that have limited the popularity of regional nerve block and infiltration anesthesia in proctologic surgery.

Numerous drugs and many technics to increase the efficiency of regional anesthetics have been tested in recent years, but Cosentino[2] and Duran-Reynals[3] were the first to suggest addition of hyaluronidase to local anesthetic solutions. Neither, however, recorded any experimental or clinical data.

Hyaluronidase, a mucolytic enzyme that hydrolyzes the mucopolysaccharide, hyaluronic acid ("ground substance" present in the interstices of the tissues), has the action of the "spreading factor" originally reported by Duran-Reynals.[4]

The rate of the diffusion is proportionate to the amount of the enzyme, and the extent is proportionate to the volume of the fluid injected.[5,6] Hechter[7] found that the natural hyaluronic acid barrier was partly restored in twenty-four hours and completely restored in forty-eight. Solutions containing hyaluronidase can penetrate a fibrin barrier only if injected into it; otherwise the drug diffuses around it and follows the path of the least resistance.[8]

Hyaluronidase has proved completely nontoxic in clinical use.[5,8–10] In animals intravenous administration of 500 times the maximal therapeutic dose causes no significant change in blood pressure, respiration, body temperature or kidney function, and no histologic changes in the tissues.[5,9,11]

In the studies of Kirby and Looby[12] on the action of hyaluronidase in local anesthesia, addition of the enzyme to the local anesthetic solution increased the area of anesthetized skin by 40 per cent.

Other investigators have demonstrated the value of hyaluronidase in local anesthesia for all types of general and special surgery.[12–19] No report has appeared, however, on the use of hyaluronidase in proctologic surgery.

In this series 357 patients were operated upon under local anesthesia produced by subcutaneous infiltration of a solution containing procaine, epinephrine or cobefrin® and hyaluronidase.* The number of patients reported in this series includes only those who were treated as inpatients in the hospital. There was, however, an additional number of patients upon whom this solution was used to advantage for minor office procedures. This method was used in all types of anorectal procedures, both major and minor. While it is not

* Wydase® (lyophilized hyaluronidase, Wyeth, Inc., Philadelphia, Pa. Wydase is measured in TR (turbidity-reducing) units. A TR unit is defined as the amount of hyaluronidase which will reduce the turbidity produced by 0.2 mg. of potassium hyaluronate in acidified horse serum to that produced by 0.1 mg. under assay conditions.

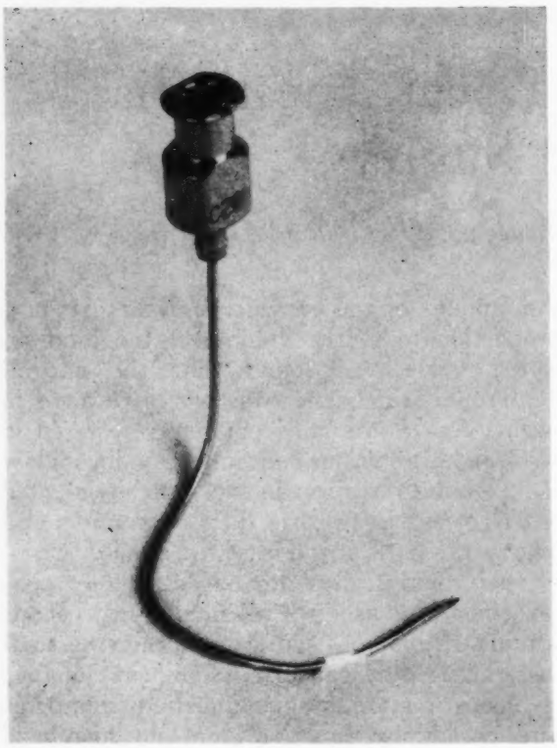

FIG. 1. Author's modification of a 22 gauge 2 inch needle for anal infiltration technic.

suggested that this technic will supplant older methods since it may not be suitable for all types of proctologic surgery, it is believed that infiltration anesthesia with the addition of hyaluronidase will prove of definite advantage in operations on the anus and lower rectum, when indicated. Its principal contraindications are the presence of active suppurative disease and possibly, in some instances, severe hypertension, although the use of cobefrin rather than adrenalin® has minimized the hypertensive effect of the solution.

Technic. Since most patients are apprehensive and approach any surgery with great fear, adequate preoperative sedation is important. Most patients in this series received 1½ gr. of pentobarbital sodium one hour before operation, followed by 1½ gr. of secobarbital in half an hour. In the majority of instances the use of postoperative sedation was minimal.

An enema was given the night before and on the morning of the operation, and after each enema the entire perianal surface was thoroughly cleansed with pHisoderm® solution which provides a long-lasting antiseptic effect.

At operation the patient was placed in the jackknife position and the buttocks retracted with strips of adhesive. The skin of the operative field was again cleansed with pHisoderm solution and an aqueous solution of zephiran®. It is important that this careful cleansing technic be followed carefully to minimize subsequent infection.

The anesthetic solution was prepared by dissolving 300 TR units of hyaluronidase in 50 cc. of 1 per cent procaine with 0.5 cc. of epinephrine hydrochloride solution U.S.P., 1:1000. Recently, a 1 per cent procaine-nordefrin hydrochloride mixture has been used in order to minimize the hypertensive effect which the epinephrine solution caused in some instances. Approximately 18 to 25 cc. of the anesthetic-hyaluronidase-epinephrine solution was sufficient for each operation.

Multiple needle punctures disturb most patients, therefore it seemed desirable to devise some method of reducing the number of injections required to provide an adequate anesthesia. Since an ordinary 22 gauge, 1½ inch malleable steel needle can be bent to any desired shape at the operating table, the idea of curving the needle to form a semi-circle promised to solve the problem of injecting the anesthetic for anorectal operations. (Fig. 1.) With a needle of this shape the anesthetic solution could be introduced satisfactorily into the involved area, and the patient would be spared the discomfort and the additional risk of injection entailed by numerous perforations of the skin. A 10 cc. Pitkin syringe may be used to advantage. In order to obtain further muscle relaxation, 3 to 4 cc. of the solution is then injected into each quadrant beneath the anoderm and lower rectal mucosa using a straight 24 gauge 1½ inch needle. The index finger is placed in the anal canal in order to determine the depth of the injection and to prevent perforation of the skin and lower rectal mucosa. With this technic it was possible to infiltrate the entire perianal region and obtain complete anesthesia of the anal canal and sphincter musculature.

Of course it is not necessary to use this technic. The ordinary method of subcutaneous infiltration in which the needle puncture is made in each lateral quadrant may be preferred by those who favor this method.

Results. Relaxation of the sphincter occurred immediately and the operation could be started without delay. This is a desirable feature obtained by this method.

Because of the addition of epinephrine or cobefrin to the anesthetic there was very little bleeding during the procedure. Hence, the anatomy of the anorectal region was more clearly delineated and as a result of the drug action cleavage planes were more readily discernible, enabling a finer operative technic. Little or no distortion of the tissues occurred following the injection of the solution.

Analgesia produced by this method equaled the effects of anesthetics administered by the spinal or caudal routes and certainly was much more satisfactory than the results to be expected with inhalant agents ordinarily used. Duration of anesthesia was also of advantage in this series; analgesia lasted as long as two to four hours following the operation.

Where a great deal of perianal edema existed it was found that the hyaluronidase caused its complete disappearance within thirty seconds following injection. This allowed for improved technic, less trauma to the tissues and removed the risk of excising more than the necessary amount of skin. It also allowed for a much earlier operation in those patients who had developed hemorrhoidal prolapse with marked edema. Where prolapse existed, its extent was more easily determined by having the unanesthetized patient consciously express the prolapse.

In several instances results were less satisfactory than anticipated. On one occasion extreme concern was caused by a severe blood pressure elevation due to the epinephrine effect. This was quickly controlled by the immediate inhalation of amyl nitrite. As a direct result of this effect further studies indicated the presence of pheochromocytoma in this individual. However, it was at this point that cobefrin was substituted for adrenalin, and no marked blood pressure rise has been caused using this drug. One acute bilateral subcutaneous abscess resulted in this series. No other acute infection was noted in any of the patients studied. In another instance a hematoma was caused by the needle perforating an external varix. This was not considered important since the involved hemorrhoidal segment would have been excised in any event.

SUMMARY

A new technic is described for local anesthesia in operations on the anus and lower rectum. The method makes use of a curved needle and employs the spreading action of hyaluronidase to facilitate infiltration of the perianal tissues with the anesthetic solution.

Anorectal surgery was performed on 357 hospitalized patients. Relaxation of the sphincter was almost instantaneous. Analgesia equaled the results obtained with other methods of anesthesia. Postoperative discomfort was minimal. There was little or no distortion of tissues from the anesthetic solution and only one abscess was noted in the series. Where perianal edema pre-existed, the edema rapidly disappeared following infiltration with the solution.

The use of hyaluronidase enhances the efficiency of local anesthesia in anorectal procedures. As clinical experience with the described method increases, it is becoming clear that hyaluronidase merits more general use in anorectal surgery.

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Epidermoid Carcinoma of the Anus and Rectum

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EPITHELIOMA of the anus and rectum are considered rare. This paper is presented to report seven cases of epidermoid carcinoma occurring in 103 cases of proven malignancy of the anus, rectum and colon.

The usually reported sex incidence is equally divided with a slight predominance in the female. Six of the patients in this study were females and one was male. The age ranged from thirty-four to sixty years, with an average age incidence of fifty years.

The lesion may be in the anal canal, at or near the anorectal line extending either downward or up into the rectum, in the perianal region, or infrequently in the ampulla. Buie[1] has reported five cases within the rectum, well above the anorectal line.

Bacon[2] states that squamous cell carcinoma metastasizes less frequently and less widely than adenocarcinoma. Locally it is more invasive but grows more slowly. The mode of spread is by continuity (infiltration), by the lymphatics (which is most important) and by the blood stream. Local invasion of the sphincter musculature, perianal tissues, rectovaginal septum or prostate is not uncommon. Bacon further states that lymphatic metastasis depends upon the location of the lesion; he divides the lymphatics into two main groups, namely, the inferior and superior. The inferior group, situated below the anorectal line, drains through the perineum to the inguinal lymphatics. The superior group drains the area above the anorectal line. This group drains into the retrorectal or sacral glands finally, to reach, the preaortic glands, laterally to the obturator and then the internal iliac glands. Instances of spread into and above the anus have occurred through the intramural chain which communicates the inferior and superior group of lymphatics.

Binkley[3] revealed there were twenty-seven different symptoms reported by the patients. These fall into four main groups. The most frequent symptom encountered is the passage of blood. The second most frequent symptom consisted of distress, the severity ranging from mild discomfort to excruciating pain. The third was a change in bowel habit; the largest number of patients reported constipation or a decrease in the number of stools. The last group consisted of miscellaneous complaints such as itching, mass in perineum and the late symptoms of cancer (anorexia, loss of weight and strength, and anemia).

TABLE 1
ANORECTAL EPITHELIOMAS

Case	Race and Sex	Age	Rectal Bleeding	Pain	Mass	Constipation	Itching	Loss of Weight
I	W, F	60	x	x	..	..	x	..
II	W, F	58	x	..	x	..	..	x
III	W, F	43	x	..	..	..	..	..
IV	W, F	53	x	..	x	x	..	..
V	W, F	60	..	x	x	..	..	..
VI	W, M	53	x	..	..	..	..	..
VII	W, F	34	x	..	..	..	..	..

Six of the patients reported bleeding, while pain was the presenting complaint in three. Three patients noted the presence of a mass and one complained of itching. A change in bowel habit manifested by constipation occurred in one patient and loss of weight was present in another.

Table 1 lists a summary of symptoms in the seven cases of anorectal epitheliomas presented herein.

The definitive diagnosis can only be made on biopsy or pathologic study of all tissue removed from the anorectum. Binkley[3] has aptly stated that it cannot be made on symptoms alone. McQuarrie and Buie[4] state that all tissue excised must be examined microscopically and that all lesions which do not heal should be biopsied.

The treatment of epidermoid carcinoma is still not well standardized. Bacon[2] states that his treatment of choice is a Miles resection followed by bilateral inguinal node dissection. Moore[5] establishes a colostomy followed by intensive x-ray and radium therapy. Crile[6]

advocates a Miles resection and then waits for any indications of metastasis before undertaking bilateral node dissection. Binkley,[3] in a very thorough review of a large series of cases, states that treatment is based more on the size and location of the tumor. Small

TABLE II
METHOD OF TREATMENT

Case	Type of Treatment	Node Dissection	Prognosis
I	Thirty 1 mc. gold radon implants 1 cm. apart	Not offered	Alive 3 yr., 5 mo.; recurrence
II	Miles resection	Not offered	Alive and well 5 yr. without evidence of recurrence
III	7,000 R.U.; Miles resection	Not offered	Died 2 yr., 9 mo. with extensive recurrence of perineum
IV	Miles resection	Not offered	Died 2 yr., 11 mo. with abdominal carcinomatosis
V	Miles resection	Refused	Alive and well 1 yr., 9 mo.
VI	3,240 R.U. to perineum; Miles resection	Not offered	Alive and well 2 yr., 4 mo.
VII	Found at time of hemorroidectomy	Not offered	Alive and well 1 yr., 5 mo.

tumors below the mucocutaneous junction are treated by local excision and radiation therapy; large tumors and those situated above the anorectal line receive preoperative high voltage roentgen ray therapy and a wide one-stage abdominoperineal resection. He states that radical inguinal node dissection is necessary with positive and suspicious inguinal metastasis. Sweet,[7] in a report of seventy-seven cases treated by surgery and irradiation, concludes surgery is the most satisfactory method of treatment, either an abdominoperineal resection or colostomy and posterior resection, but he does not mention inguinal node resection.

Harvey[8] reported results of treatment in thirty-seven cases and recommended local excision of small tumors and radiotherapy, and preferably a combination of both. For large tumors with metastasis he advocates radical resection and bilateral node dissection; while in locally extensive growths, he favors temporary colostomy and radiation therapy. He further states that irradiation cannot destroy perirectal nodes.

McQuarrie, Harlow and Buie[4] reported 137 cases with various types of treatment; however, they made no conclusion as to treatment but were impressed with the necessity for biopsy of all tissue and anal wounds that will not heal.

Cattell and Williams[9] reviewed the cases at the Lahey Clinic and recommended a Miles resection followed by bilateral inguinal node dissection to include the femoral and iliac nodes.

LaBlanc, Buie and Docherty[1] reported five cases of epidermoid carcinoma within the rectum and recommended surgery. Four patients had had posterior resections and one abdominoperineal resection.

Bacon, Venturo and Sauer[10] reported twenty-eight cases and recommended a radical Miles resection with possible radium implants in the obturator and levator ani muscle stumps and bilateral inguinal node dissection.

Keyes[11] reported thirty-five cases, seventeen of which were treated either by surgery or radiation; seven were cancer-free for five years. He concluded that squamous carcinoma can be treated successfully either by surgery or radiation.

The treatment varies with different investigators as well as in our seven cases. (Table II.)

The survival rate was not good, with two dead from local recurrent carcinoma in less than three years postoperative, one with a known recurrence but still alive, one surviving five years cancer-free and the other three not having sufficient postoperative time to be of any value in determining their prognosis even though there is no evidence of recurrence at this time.

Cases I, II, IV and V had grade III epidermoid carcinoma. Case VI had a rather unusual sequence of events in that low-grade carcinoma (early invasive squamous cell carcinoma, grade II) was found on routine fistulectomy and hemorrhoidectomy. Following 3,240 R.U. of radiation therapy the epithelioma had not been destroyed and biopsy revealed low-grade epidermoid carcinoma. Because of the presence of cancer cells an abdominoperineal resection was performed and careful microscopic study revealed diffuse leukoplakia involving anal canal and perianal skin, with a few atypical epithelial changes. Case VII is of interest because non-invasive epidermoid carcinoma was found on routine hemorrhoidectomy.

SUMMARY

Only one patient of the seven cases studied is apparently cancer-free for five years, a survival

rate of 14 per cent. However, if the patient with known recurrence given only palliative radium is excluded, the survival rate would be 16.6 per cent. The outcome of the remaining three patients who are apparently free of this disease from a minimum of one year, five months to two years, four months may help increase the survival rate.

The rather high incidence of epidermoid carcinoma, 6.7 per cent, occurring in this series of cases is rather unusual. If all tissue removed from the anorectum is submitted for pathologic study and a biopsy is obtained of all suspicious lesions, an earlier diagnosis will be established. Radical exterpation seems to be the treatment of choice. If the question of routine bilateral node dissection with its associated increased morbidity can be analyzed and if a more thorough study of the results from pre- or postoperative irradiation therapy can be obtained, there may be some hope of increasing the survival rate for these patients.

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Bowen's Disease of the Anal Region (Squamous Cell Carcinoma *in Situ*)

REPORT OF THREE CASES

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BOWEN's disease is a chronic, intraepidermal, squamous cell epithelioma well known to dermatologists. It is usually found on the glabrous skin, but mucosal and conjunctival lesions have been reported.[4,5,11,15] The reported occurrence of this lesion in the anal region has been infrequent.[1,6,11,20] During the past fourteen years only two papers dealing with anal lesions have appeared in the literature.[6,11] One of these was based on a case report given before the American Proctologic Society in 1941 by Freund.[6]

A concise description of Bowen's disease was presented by McCarthy in 1931.[12] He described it as "an affection of long standing characterized clinically by the formation of single or multiple, papulosquamous, eroded or crusty, hyperkeratotic tumorlike lesions of the skin. Exceptionally, the lesions may take the form of the flat, plateaulike cellular infiltrations of a true papillomatous form. The lesions, no matter what the clinical form, may have the power of invading the lymph spaces and glands and of producing metastasis in the inner organs, although as a rule they are benign and do not form metastasis."

Bowen titled his original papers on this disease in 1912 and 1915 "precancerous dermatoses" and yet in his third reported case metastatic axillary lymph glands developed.[2,3] Later authors have continued this concept and have included Bowen's disease, Paget's disease, Queyrat's erythroplasia and others in a group classified as the precancerous dermatoses.

A more modern and realistic concept considers Bowen's disease from the outset as a slow-maturing, intraepidermal squamous cell carcinoma *in situ*, separated from the dermis by an intact basal cell layer.[7,18,19,20] An analogy can be made to cancer *in situ* in other regions, such as an intraepithelial cancer of the cervix uteri or one at the tip of an adenomatous rectal polyp where the basement membrane limits spread. Squamous cell carcinoma *in situ* is the more suitable term to describe the preinvasive phase of the tumor. When Bowen's epithelioma penetrates the dermal layer, it acts as a grade II or III squamous cell cancer prone to infiltration and metastasis, and never as a basal cell epithelioma.[8,10,18]

CLINICAL CONSIDERATIONS

Bowen's tumor is usually found in the over forty age group and is common to both sexes. The clinical diagnosis of an anal Bowen's epithelioma is extremely difficult to establish solely from the surface appearance of the lesion. This chronic condition may remain static for years, but will eventually undergo varied surface changes which often mimic other skin lesions. The clinical differential diagnosis will include early stages of basal cell epithelioma, varied keratoses and dermatitis, eczematoid conditions, psoriasis, luetic lesions, condylomas and extramammary Paget's disease.[7,13,14,16,17]

The chronicity of the lesion with the marked tendency to remain intraepidermal for years before invading is indeed fortunate for treatment and prognosis. Operatively, an excision of Bowen's tumor with normal skin margins will suffice for cure if the basal cell layer is found intact on serial section.

PATHOLOGY

Histologic examination of the entire Bowen's lesion is the method by which an accurate diagnosis and prognosis can be made. The microscopic picture, unlike the inconstant and varied gross surface lesion, is fairly typical and diagnostic.

The basic lesion is a hyperplasia in the malpighian layer of the epidermis which exhibits a generally disordered appearance. Characteristic is the Bowen cell, a large atypical cell with haloed giant or "clumped" hyperchromatic nuclei produced by amitotic cell division. Malignant dyskeratoses with individual cell keratinization and epithelial pearl formation may be seen in the prickly layer, simulating that found in a more dynamic type of squamous cell cancer. Mitotic and amitotic figures are often numerous. Intracellular edema produces a haloed effect on the large hyperchromatic nuclei of the atypical cells. The intercellular prickles of the malpighian layer are always retained. Generally, the picture is a proliferation and acanthosis in the epidermis with variable mixtures of dyskeratosis, parakeratosis and hyperkeratosis.[7,10,12,15]

The basal cell layer is usually intact; but if invasion into the dermis has occurred, the probability of metastasis is great.[10,18] The papillary portion of the dermis invariably shows a chronic inflammatory reaction, evidenced by round and plasma cell infiltrations. Extramammary Paget's disease may resemble Bowen's epithelioma histologically, but there is a loss of prickles around the haloed Paget cells and intercellular edema is marked in Paget's disease.[10,14,17]

In recent months the author has had the opportunity of observing three patients with anal Bowen's disease. These are being presented because of the rarity of such cases in the literature.

CASE REPORTS

CASE I. E. F., a fifty-three year old white married male realtor, was seen on January 14, 1953, complaining of protruding anal masses and soreness after evacuation for several months. He had also noticed a painless lump of constant size near the anus for over three years, which gave him no concern. The history was otherwise negative. Proctology examination revealed a firm, ovoid, painless, brownish tumor with a warty surface, 1.5 by 2 cm., in the left anterior perianal region. Large prolapsing epithelized internal hemorrhoids, external tags and a large prolapsing fibrous anal papilla were noted. The preoperative diagnosis was perianal papilloma and hemorrhoids. Hemorrhoidectomy was performed and the perianal tumor excised, with ample skin margins. The specimen was sent to the laboratory for study and the pathologic diagnosis was perianal Bowen's disease (squamous cell carcinoma *in situ*). (Figs. 1 and 2.)

Microscopically, the lesion presented a sharply defined zone of acanthosis, characterized by marked thickening and elongation of the rete pegs. The stratum malpighii was composed of cells with large coarsely granular basophilic nuclei in a scant cytoplasm. Mitoses were frequent. In some areas there were malignant dyskeratoses and large bizarre cells with "clumped" and giant haloed nuclei. The basal layer was uniform and intact. A plasma and small round cell infiltration was noted in the subjacent dermis.

CASE II. A. E. W., a sixty-seven year old unmarried white woman, was hospitalized February 16, 1953, for an asymptomatic plaque-like ulcerating perianal growth of over five years' duration. The lesion had been seen by several physicians but a definite diagnosis had not been made. The history was otherwise negative except for moderate obesity and hypertension. Proctology examination showed an eroded, red, plaque-like ovoid patch without induration or tenderness, at the right anterior anal margin. The preoperative diagnosis was perianal growth, type undetermined. The tumor was removed *en bloc* with ample skin margins. The gross specimen, a spindle-shaped strip of skin, 3 by 2 by 1.2 cm., with a light red central granulating area, 1.5 by 1 cm., was sent to the pathology department. The report was squamous cell cancer *in situ* of a Bowen type. (Figs. 3 and 4.)

Microscopically, the epidermal layer showed a sharply defined zone of acanthosis with elongated branching rete pegs. The malpighian cells were in a disorderly arrangement and had large irregular basophilic nuclei. A number of small horn cysts and pearls were present. Multinuclear vacuolated cells and atypical mitoses were infrequent. Ulcerated granular areas were prominent and the subjacent corium showed a marked round and plasma cell infiltration.

CASE III. O. A. F., a forty-seven year old white housewife, entered the University of California Hospital October 27, 1953, complaining of persistent anal protrusions following surgery for vulvar leukoplakia three years previously. General health was good except for considerable vaginal discharge and an irritated

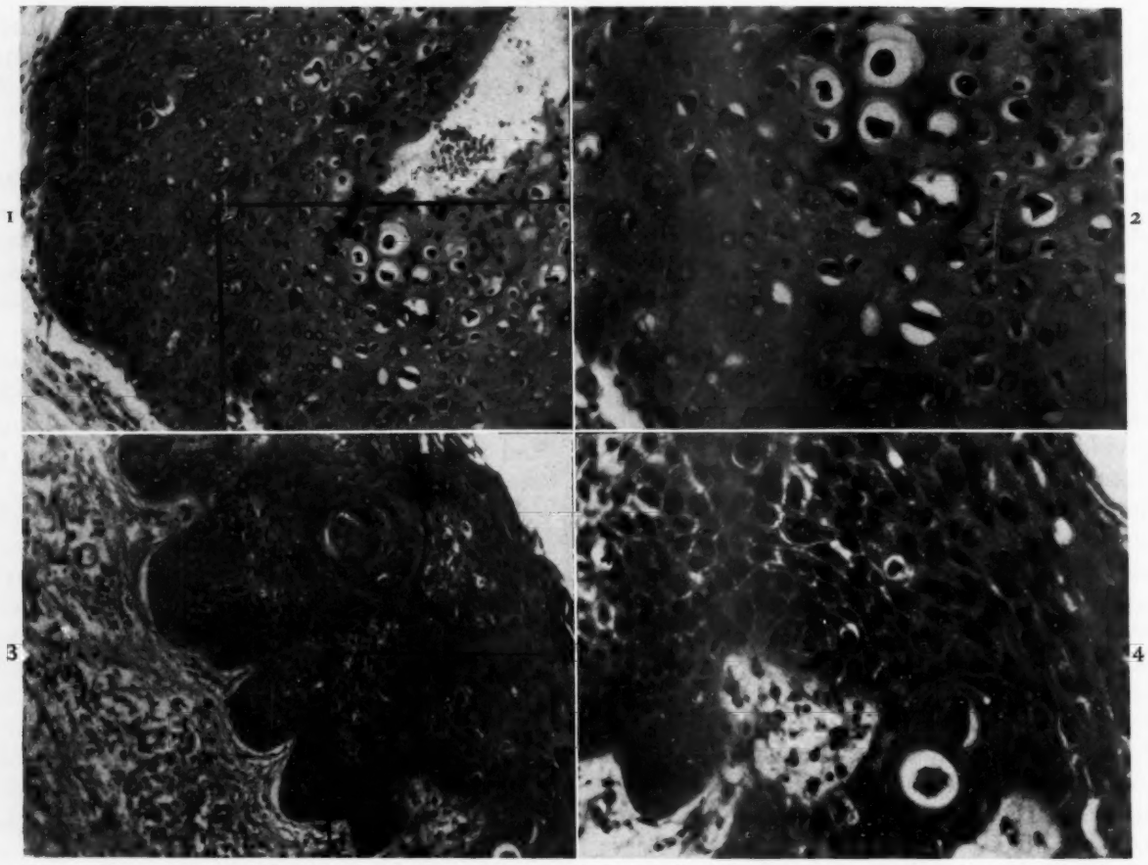

FIG. 1. Case I. Section showing a thickened acanthotic epidermis; disorderly hyperplasia of the malpighian layer; parakeratosis and an intact basal layer. Dyskeratosis is present; × 160.

FIG. 2. Case I. Epidermis showing characteristic haloed Bowen cells with hyperchromatic giant and "clumped" nuclei; × 320.

FIG. 3. Case II. Section showing acanthosis and epithelial pearl formation in the malpighian layer. The basal layer is intact and the subjacent dermis shows a marked chronic inflammatory infiltrate; × 160.

FIG. 4. Case II. Epidermis shows malignant dyskeratoses with individual cell keratinization and horn pearl formation; × 320.

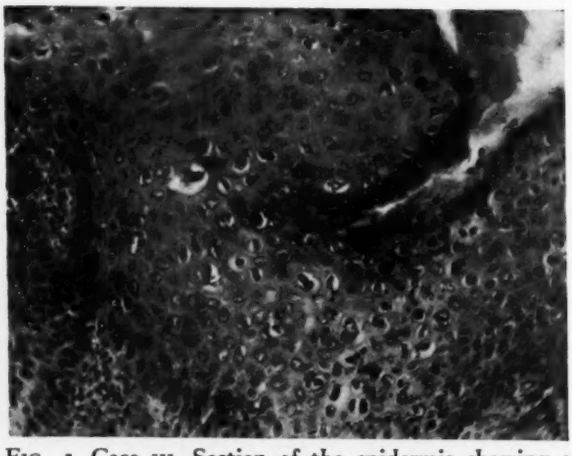

FIG. 5. Case III. Section of the epidermis showing a disorderly pattern in the malpighian layer. Many cells have large irregular hyperchromatic nuclei with perinuclear edema. Mitotic figures are common; × 160.

edematous vulvar skin. Proctology examination showed two large external anal condylomatous skin tags, 1.5 by 1.5 cm. at 3 and 9 o'clock, and a smaller tag posteriorly. The preoperative diagnosis was anal condylomas. External hemorrhoidectomy was done with the Bovie unit and the tissue sent to the pathology department for examination. The pathologic diagnosis was condyloma acuminata with squamous cell carcinoma *in situ* (Bowen's disease). (Fig. 5.)

Pathologically, the gross specimens consisted of pieces of skin and subcutaneous tissue studded with multiple small broad-based papillary tags. Microscopically, the epidermis showed a marked parakeratosis with nucleated keratin cells and acanthosis of the rete pegs.

Some of the malpighian cells had large irregular hyperchromatic nuclei with perinuclear edema. Mitotic and amitotic figures were common. The basal layer consisted of uniform cuboidal cells. A lymphocytic infiltration was present in the subjacent dermis.

COMMENT

Each of the three reported cases had a clinically different type of anal lesion of more than three years' duration. Excision of the entire tumor with adequate skin margins and study of representative serial sections established the diagnosis of squamous cell cancer *in situ* of the Bowen type. The absence of dermal invasion was determined by microscopy and indicated a favorable prognosis. These tumors can be catalogued as a type of epidermoid carcinoma of less than grade 1 malignancy based on a lower ratio of atypia and the absence of invasion. Roentgen therapy or fulguration is inadvisable in the treatment of anal Bowen's epithelioma.

SUMMARY

1. Bowen's disease of the anal region is a rare chronic and clinically varied type of intraepidermal tumor with infiltrating and metastatic potentia. Squamous cell cancer *in situ* is a more proper classification than precancerous dermatoses.

2. The diagnosis of a Bowen's epithelioma is made by histologic examination of the tissue. Serial section with careful examination of the entire tumor is required to eliminate the possibility of invasion.

3. Local excision offers a good prognosis because of the tendency of Bowen's epithelioma to remain intraepidermal for many years before penetrating the dermal layer.

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DISCUSSIONS

Edward L. Clanton (Spokane, Wash.): Dr. Grodsky's paper was very complete and leaves little to be said on Bowen's disease itself. I think he makes a very good point in that he considers this a carcinoma *in situ*. Certainly it is a malignant lesion since it may destroy the patient; however, it is not highly malignant. A lesion such as this should be stressed before a group of men who are seeing the area in which this lesion may be found.

Now, to me, it is a very rare lesion as I have never seen one although I have talked with men here who have. After the publishing of this paper I am sure there will be more cases in the literature. It does bring up a point, which, is that all tissue taken from the anorectal region at the time of surgery should be sent for examination.

It is up to us, as surgeons, to see that this is done and to designate the areas that are suspicious. Before we can do this we must keep the infrequently seen and rare lesions in mind.

Albert G. Schutte (Milwaukee, Wisc.): Last year I found two small lesions less than

the size of a kernel of rice in this area and lesions not that thick in two other patients with pruritus ani. They did not look right so I resected them elliptically and the pathologic report was squamous cell carcinoma *in situ*. I would like to ask Dr. Grodsky whether these could be classed as Bowen's disease.

LEWIS GRODSKY (closing): In answer to Dr. Schutte's question I would say that Bowen's disease is squamous cell carcinoma *in situ* of a certain type in which large atypical Bowen cells with haloed giant or "clumped" hyperchromatic nuclei are found. It may be a misleading classification. As a matter of fact, in reporting this rare lesion our pathology department at the University of California will not classify it as Bowen's disease. They prefer to call it carcinoma *in situ* and that is exactly what it is.

You may wonder about the degree of malignancy of Bowen's type of carcinoma *in situ*. I would say if you want to use Broders' system you might call it grade ¾, but it is certainly under a grade 1 malignancy.

The other point I wish to bring out is the possibility of invasion in this type of tumor. Exact figures are unknown except for a paper written in 1939 by A. B. Stout on the malignant manifestations of Bowen's disease. He states that in the equivalent mucosal lesion the possibility of metastases is 40 per cent while in the skin lesion discussed here it is between 2 and 3 per cent. Montgomery cites cases in which the lesions have remained *in situ* for twenty years or more. This condition is not a precancerous dermatosis but is a squamous cell carcinoma *in situ* with an invasion index of 2 or 3 per cent.

Retrograde Rectal Hemorrhage

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IT is the purpose of this paper to call attention to two facts: (1) That blood discovered in the sigmoid and upper rectum on initial proctoscopic examination may originate at or near the mucocutaneous line and be carried proximally by retrograde filling, reverse peristalsis or by the passage of the examining instrument and (2) that serious rectal hemorrhage may originate at or near the mucocutaneous line.

During proctoscopic examination in some patients it has been observed that a blue, green or orange colored material may be seen on the mucosa as high as the 25 cm. level. Such patients give a history of having used a so-called hemorrhoidal suppository a few hours prior to the examination. Since a suppository upon insertion lies just above the mucocutaneous line, the colored material is carried proximally by a physiologic mechanism. Presumably, this process is a reverse peristalsis.

To confirm this observation commercial, so-called hemorrhoidal suppositories were inserted in twenty patients, who were then asked to return in one hour. Proctoscopic examination was then carried out. The examination was done in an uninverted position to avoid the effect of gravity, and the lower rectum was swabbed until clean to avoid carrying the colored material proximally with the proctoscope. In ten patients the colored material was found in the rectum and sigmoid; in three it stained the rectal mucosa up to the rectosigmoid where a fecal mass was encountered; in four the discoloration was confined to the ampulla by fecal material, and in three the partially melted suppository was found just above the mucocutaneous line. These results seem to indicate that a reverse peristalsis may be involved.

A group of ten patients were examined one hour after the insertion of 10 cc. of whole blood into the rectum. During the waiting period they were asked to sit or stand, but not to recline in order to avoid the effect of gravity. On proctoscopic examination the lower rectum was swabbed until clean and then the bowel was examined proximally. In seven cases small amounts of light and dark blood, clotted or streaked, were found up to the 25 cm. level. In three cases fecal material in the ampulla blocked any retrograde flow. From this preliminary study it would seem that blood also may be carried proximally by reverse peristalsis.

Another group of ten patients, each with bleeding internal hemorrhoids, were asked to have their bowel movement in the office lavatory. They were examined within the next thirty minutes. In seven patients there was no blood found in the rectum although six of these passed some blood with the bowel movement. In three patients small amounts of blood were seen in the ampulla. After cleaning away this blood, proctoscopic examination was continued, and in one case blood was seen at the 20 cm. level. These results may indicate that in a small but definite percentage of patients with bleeding internal hemorrhoids, some residual bleeding occurs after defecation. This may pass unnoticed by the patient or appear as a dark stain on the distal end of the next fecal passage.

The proctoscope may also carry blood proximally. The hollow tip of the instrument may pick up the blood below and deposit small amounts of it along the mucosa to the 25 cm. level. This blood may originate, as above described, from digital examination in hemorrhoids, but most commonly from slight injury to the anterior bowel wall by the obturator of the anoscope or proctoscope. The bleeding from such injury may be slight and unnoticed. The obturator of the anoscope may scratch the mucosa, and being longer than the examining part of the instrument cause a little bleeding which is not seen during anoscopic examination. The proctoscope is then passed and before its obturator is withdrawn the proximal end of the instrument may have passed over the site of the injury and have picked up a little blood. The blood may remain unseen until the instrument is rotated, or a cotton applicator is

passed or when intestinal mucus picks it up from the instrument. This may occur at any level but it is probably more likely to occur in the sigmoid because of the relatively narrow lumen. The obturator of the proctoscope may itself injure the bowel enough to cause slight bleeding which may be carried proximally.

TABLE 1

TABULATION OF CASES OF RECTAL HEMORRHAGE ORIGINATING AT OR NEAR THE MUCOCUTANEOUS LINE

Case No.	Diagnosis	Treatment	X-ray	Further Bleeding
1	Submucosal abscess at 4 cm.	Fulguration; surgery	Neg.	None, 2 mo.
2	Trauma by enema tip, 5 cm.	Fulguration	Neg.	None, 42 mo.
3	Trauma by enema tip, 6 cm.	Pressure	Neg.	None, 30 mo.
4	Injection slough	Fulguration	Neg.	None, 6 yr.
5	Injection slough	Fulguration	Neg.	None, 31 mo.
6	Internal hemorrhoids	Symptomatic	Neg.	None, 1 mo.
7	Internal hemorrhoids, thrombotic	Surgery	Pending	None, 6 mo.
8	Internal hemorrhoids	Transfixion; injections	Neg.	Slight, 4 wk.; none, 2 mo.
9	Adenocarcinoma at 3 cm.	Fulguration; biopsy and abdominoperineal resection	Reported neg.	Unknown
10	Adenocarcinoma at 10 cm.	Fulguration; biopsy and abdominoperineal	Neg.	None, 2 yr.
11	Distended veins; cirrhosis	Surgical ligation	Neg.	None, 1 yr.
12	Hypertrophic papilla	Surgery	Neg.	None, 4 mo.
13	Hypertrophic papilla; hemorrhoid	Surgery	Neg.	None, 6 yr.
14	Foreign body	Removal; fulguration	Refused	None, 3 mo.
15	Factitial proctitis	Symptomatic	Neg.	Slight 5 wk.; none, 3 yr.
16	Factitial proctitis	Symptomatic	Neg.	Slight 7 wk.; none 4 yr.
17	Internal thrombosis	Symptomatic	Neg.	None, 5 yr.
18	Bacillary dysentery	Medical (elsewhere)	None	Reported none, 5 yr.
19	Ulcerative colitis	Medical (elsewhere)	Neg., later ulcers	Continued 6 mo.; now reported in remission
20	Spontaneous extrusion; polyp	Fulguration	Neg.	None, 5 yr.
21	Spontaneous extrusion; polyp	Fulguration	Neg.	None, 2 yr.
22	Bleeding at anastomosis	Fulguration	Neg.	None, 2 yr.
23	Bleeding at anastomosis	Fulguration; biopsy	See text	None, 1 mo.
24	Postoperative hemorrhoidectomy	Fulguration	Refused	None, 4½ yr.
25	Postoperative hemorrhoidectomy	Ligation	Neg.	None, 8 yr.

To illustrate that serious rectal hemorrhage may originate at or near the mucocutaneous line twenty-five cases are tabulated. (Table 1.) The causes of the bleeding include the following; submucosal abscess, trauma by enema tip, injection slough, internal hemorrhoids, adenocarcinoma, distended veins secondary to cirrhosis hypertrophic papillae, foreign body, factitial proctitis, internal thrombosis, bacillary dysentery, ulcerative colitis, spontaneous extrusion of a polyp, ulcer at anastomosis and postoperative hemorrhoidectomy. In each case the cause of the bleeding was discovered at or near the mucocutaneous line, although a varied amount of blood and clots was seen at the higher levels. The lesions were treated or removed surgically, and in all but four cases active bleeding stopped immediately although residual dark blood was passed in gradually lessening amounts for a day or two. In the two cases of factitial proctitis slight bleeding continued for a few weeks. The patient with ulcerative colitis continued to have some bleeding for six months and one of the patients with internal hemorrhoids had some bleeding with defecation for four weeks. Barium enemas with air contrast studies were done in twenty cases and reported negative in nineteen. Case 23 showed a distortion of the bowel above an anastomosis; in two cases x-rays were taken elsewhere; two patients refused x-rays, and in one it is pending.

The extent of the hemorrhage varied from a cupful to 1,500 cc. The constitutional symptoms varied from none to moderately severe shock. Eight of the lesions were palpable. Five were visible on initial anoscopic examination, six were visible anoscopically only after cleansing of the bowel and fourteen were not visible anoscopically at all. Four lesions were seen by the proctoscope before aspiration and cleansing of the bowel, fifteen were seen proctoscopically only after cleansing of the bowel and six were not visible proctoscopically.

When patients appear following rectal hemorrhage, the clinician may be inclined to do a proctoscopic examination at once. A digital and anoscopic examination should be done first. If the result of the anoscopic examination is unsatisfactory because of the presence of a

large amount of blood in the lower rectum, it should be repeated after proctoscopy is done and the bowel cleaned out. The importance of anoscopy is stressed because patients may have a lesion in the rectum and some demonstrable disturbance at the mucocutaneous line (Case 10). It also extablishes the diagnosis in very low-lying lesions such as bleeding hypertrophic papillae or injection sloughs[1] which may be missed by the proctoscope. When the latter instrument is inserted and before visualization begins, the proximal end of it passes over an area of 2 to 3 cm. of the lower rectum; this area is seen only briefly on withdrawal of the instrument. Some of the reported lesions were in this area.

As to how the blood reaches the sigmoid in these cases, it may be assumed that it is either by retrograde filling or reverse peristalsis. Preliminary experiments with the instillation of whole blood into the rectum seem to indicate that amounts of 30 cc. or more in most cases causes a desire to defecate. Therefore, in slowly bleeding lesions defecation may occur before retrograde filling could bring the blood up to the sigmoid. In these cases reverse peristalsis may be the mechanism. In rapidly bleeding lesions the mechanism is probably a combination of retrograde filling and reverse peristalsis.

It cannot be overemphasized that these observations are in no way meant to suggest a relaxation in the careful search for neoplasms of the bowel. The coexistence of low rectal and anal lesions with carcinoma and polyps higher up is common enough to make vigilant search mandatory. In these cases, however, bleeding was discovered on initial examination only, and was followed by discovery and treatment of the lesion and subsidence of symptoms. Barium enema, x-ray and air contrast studies were employed. If bleeding were to continue, even in the presence of negative x-rays, good judgment would call for repeated x-ray examination of the entire gastrointestinal tract and repeated proctoscopic examination. On the other hand, low rectal and anorectal lesions are not ordinarily visualized on x-ray examination. They may be missed unless a thorough examination is done including digital, anoscopic and proctoscopic examination, and adequate cleansing of the bowel by means of swabbing, irrigation and suction. Proctoscopy alone, resulting in the finding of a rectum full of blood, may cause the less experienced to assume that the hemorrhage originates above the 25 cm. level and much blood and time may be wasted.

SUMMARY AND CONCLUSIONS

1. Blood discovered in the sigmoid and upper rectum on initial proctoscopic examination may originate at or near the mucocutaneous line. Examination after the insertion of suppositories, the insertion of whole blood, and in cases of bleeding internal hemorrhoids after defecation, seems to indicate that a reverse peristalsis in one mechanism is involved. Retrograde filling and the passage of the examining instrument may also be involved.

2. Serious hemorrhage may originate at or near the mucocutaneous line. On examination the rectum and sigmoid may be full of blood and the lower lesion may be obscured. Twenty-five such cases are tabulated.

3. To avoid carrying blood proximally with the examining instruments, care should be used to avoid trauma by digital anoscopic and proctoscopic examination. Evidence of traumatic bleeding should be looked for on the examining finger and in the low rectum. Any traumatic bleeding should be swabbed away before proceeding, and in all cases the examination should be repeated.

4. In the examination of all cases of rectal hemorrhage the bowel should be cleansed, a digital, anoscopic and proctoscopic examination should be done and barium enema and air contrast studies should be made.

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Diverticulitis of the Cecum

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SINCE the achievement of very low mortality and morbidity rates for surgery of the colon during recent years, some diseases of this organ treated largely by conservative measures in the past should, in our opinion, be re-evaluated. This concept applies essentially to those inflammatory lesions characterized by recurrent acute episodes and prolonged disability. Cecal diverticulitis, like its sigmoid counterpart, seems to fall into this category.

This condition has been considered to be rare, and although in all probability many cases are not reported, it is only rarely discovered at autopsy. There are undoubtedly many patients with signs of an acute inflammatory process in the right lower quadrant who have been treated conservatively for one reason or another and probably some of these have had inflamed diverticula of the cecum.

Diverticula may occur throughout the intestinal tract but are by far most common in the colon. There are two types: acquired and congenital. The former are very frequently encountered in this organ and appear to represent a progressive degenerative process which results in herniations of the mucosa and submucosa through defects in the muscular coat. This variety is decidedly more common in the left colon, particularly the sigmoid. Congenital diverticula are true evaginations of the gut wall and contain all layers. These are best represented by Meckel's diverticulum of the small intestine, and by those which occur so frequently in the duodenum and occasionally in the cecum. It is the latter group with which this paper is concerned.

Certainly acquired diverticula occur in the cecum as well as the congenital type. However, most cases reported indicate the presence of a solitary diverticulum and commonly these have been found to be of the congenital variety. It is said that these diverticula are most apt to occur on either the lateral or anterior walls of the cecal pouch at about the level of the ileocecal valve.[1] According to Schnug,[2] the dependent portion of the organ near the base of the appendix is the third most common location.

Evaluation of Case Studies. Since 1928 we have encountered eighteen cases of acute cecal diverticulitis in the Straub Clinic. The first six of these cases were reported by Burgess[3] in 1939, and the balance have been summarized with a review of the more recent literature. (Table II.) Case reports up to 1947 have been well surveyed by Anderson,[4] who evaluated ninety-six cases and added eight from the Mayo Clinic files. He found this condition to occur between thirty-five and forty-five years of age, with equal frequency in men and women. As expected the diagnosis was established at the operating table 84 per cent of the time when patients were operated upon for either acute appendicitis or for the occurrence of a mass involving the cecum, grossly indistinguishable from carcinoma. All of the deaths in this series occurred in the preantibiotic period, prior to 1939.

Eighty-nine per cent of this group had been treated by some type of excision; the balance received either simple drainage or no operative treatment. The over-all mortality rate was 6.4 per cent. Two of fifty-three patients died following diverticulectomy and three of thirty-two patients died following right hemicolectomy or cecectomy. Follow-up information relative to recurrence of prolonged disability in those cases treated by simple drainage, exploration with or without excision, or in those treated without operation was lacking in this study. Anderson logically concluded at the time that diverticulitis remained a surgical disease which the operating surgeon was obliged to treat by the most definitive low risk procedure available. The excellent summary by Lauridson and Ross[1] reviewed all cases reported to that time and many of the cases reported in this study were included in their summary.

It has occurred to us that critical study of recent case reports with respect to propriety of surgical treatment might be of interest. Accordingly, the literature from 1946 to 1953

TABLE 1

Author	Year	Age (yr.)	Sex	Preoperative Diagnosis	Gross Appearance at Operation	Operation	Postoperative Course	Long-term Follow-up
Fairbank and Rob[6]	1946	50	F	Acute appendicitis; recurrent previous attacks	Indurated mass; carcinoma	Hemicolectomy	Satisfactory	?
	1946	38	M	Previous attacks of indigestion; acute appendicitis	Indurated mass; carcinoma	Hemicolectomy	Satisfactory	Remained well
Beck and Hopkins[7]	1947	12	F	Acute appendicitis	Solitary indurated nodule	Diverticulectomy	Satisfactory	?
	1947	65	M	Acute appendicitis	Inflamed mass	Cecectomy	Satisfactory	?
	1947	40	M	Acute appendicitis	Inflamed mass	Cecectomy	Satisfactory	?
	1947	21	F	Acute appendicitis	Localized indurated nodule	Diverticulectomy	Satisfactory	?
	1947	50	M	Appendix out; cholecystitis	Inflamed nodule	Diverticulectomy	Satisfactory	?
Levine[8]	1949	31	F	Acute appendicitis	Indurated inflamed mass	Diverticulectomy	Satisfactory	?
Migliaccio[9]	1946	52	F	Acute appendicitis	Localized inflamed nodule	Sulfasuxidine®; no resection; drainage only	Satisfactory	Barium enema confirmation of diverticulitis
	1946	54	M	Acute appendicitis	Indurated mass; ? carcinoma	Hemicolectomy	Satisfactory	?
	1946	26	F	Acute appendicitis	Indurated mass; ? carcinoma	Hemicolectomy	Satisfactory	?
	1947	30	M	Acute appendicitis	Inflamed mass; other cecal diverticula present	No operation	Recovery	?
	1947	32	M	Acute appendicitis	Inflamed mass	Diverticulectomy and ileostomy	Acute cholecystitis during postoperative course	?
Henry[10]	1946	25	M	Acute appendicitis	Inflamed mass	Diverticulectomy	Satisfactory	Barium enema; no other diverticula found
	1947	35	M	Acute appendicitis	Inflamed nodule	Diverticulectomy	Abdominal wall; hematoma or serum pocket	?
	1947	30	M	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	Barium enama showed other diverticula of cecum and associated colon
Kazmierski[11]	1950	37	M	Previous appendectomy; postoperative adhesions	Chronic localized inflamed reaction of cecum	Diverticulectomy	Satisfactory	?
Cangelosi and Khedroo[12]	1949	55	F	Acute appendicitis	Inflamed mass	Exteriorization	Satisfactory	?
Costin and Gaston[13]	1947	67	F	Deformation of the cecum; cause?	Paracecal mass; ? carcinoma	Hemicolectomy	Phlebothrombosis of lower extremity with recovery	Remained well
	1948	63	M	Acute appendicitis	Inflamed nodule	Diverticulectomy	Uneventful	?
Levin[14]	1950	41	M	Acute appendicitis	Inflamed mass; ? carcinoma	Hemicolectomy	Satisfactory	Remained well
Byrne, Kallan and Bassett[16]	1950	33	M	Acute appendicitis	? carcinoma	Hemicolectomy	Recovery	?
	1950	50	M	Acute appendicitis	Inflamed mass; appendix normal	Appendectomy	Satisfactory	?
	1950	24	F	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	?
	1950	25	F	Acute appendicitis	Inflamed mass; ? carcinoma	Hemicolectomy	Satisfactory	?
	1950	34	M	Appendix out; intestinal obstruction	Inflamed nodule	Diverticulectomy	Satisfactory	?
Kron and Specter[15]	1945	46	F	Acute appendicitis	Inflamed mass; ? carcinoma	Hemicolectomy	Satisfactory	?
	1948	28	F	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	?
	1949	38	F	Acute appendicitis	Inflamed nodule	Diverticulectomy	Abscess with prolonged drainage; phlebothrombosis and pulmonary embolism	?
	1949	44	F	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	?
Collins[17]	1948	46	F	Acute appendicitis	Inflamed mass	Hemicolectomy	Satisfactory	?
Newton[18]	1944	41	F	Acute appendicitis	Inflamed mass	Hemicolectomy	Satisfactory	?
	1948	44	M	Acute appendicitis	Localized inflamed nodule	Diverticulectomy	Satisfactory	?

TABLE I (*Continued*)

Author	Year	Age (yr.)	Sex	Preoperative Diagnosis	Gross Appearance at Operation	Operation	Postoperative Course	Long-term Follow-up
Shaw and Siegler[19]	1949	42	F	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	?
Reid[20]	1950	30	F	Carcinoma of cecum	Inflamed indurated mass	Hemicolectomy	Satisfactory	Remained well
	1950	70	M	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	Remained well
	1950	20	M	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	Remained well
VanMeter and Floyd[21]	1948	21	F	Acute appendicitis	Inflamed nodule	Inversion of diverticulum	Satisfactory	Well after 2 yr.; barium enema normal
Kirkman[22]	1948	25	F	Acute appendicitis	Extensive inflamed indurated area	Mikulicz resection	Prolonged recovery period	?
Geist[23]	1941	33	M	Acute appendicitis	Inflamed mass	Diverticulectomy	Wound infection	?
	1952	7	F	Acute appendicitis	Inflamed nodule	Diverticulectomy	Satisfactory	Well after 1 yr.
	1947	48	F	Acute appendicitis	Inflamed mass; ? carcinoma	Ileocolostomy; hemicolectomy in stages	Prolonged recovery	Well after 5 yr.
Lauridson and Ross[1]	1949	23	F	Acute appendicitis	Acute inflamed diverticulum with fecalith	Diverticulectomy	Satisfactory	?
	1949	29	M	Acute cholecystitis	Firm; inflamed	Right hemicolectomy	Wound sepsis; prolonged drainage	?
	1950	42	M	Acute appendicitis	Firm mass, wall of cecum	Diverticulectomy	Satisfactory	?
	1951	31	M	Acute appendicitis	Indurated mass, wall of cecum	Diverticulectomy	Satisfactory	?

has been reviewed. During the period fifty-eight cases, including the author's twelve (Tables I and II), managed by twenty different physicians have been reported. This group is comparable to those reported prior to 1947 by Anderson except for management from the time of the diagnosis. Obviously two improvements in treatment have been available for the recent group: (1) antibiotics of greater potency and broader range of effect have been available; and (2) improvement in supportive treatment generally with coincident reduction and morbidity and mortality for all surgical treatment of colon disease has been attained.

For the recent group the preoperative diagnosis was acute appendicitis in fifty-one, or 90 per cent, and a preoperative diagnosis of diverticulitis was suspected in five who had had previous appendectomies. The diagnosis of malignancy was made either before or at operation in fourteen cases.

Of the operations performed hemicolectomy was carried out in twenty-five instances, which included two multiple stage procedures and one Mikulicz resection. From these case reports indications for the extensive operations seemed to be either inability to differentiate the lesion from carcinoma, or a location close to the ileocecal valve, although one of our own cases was submitted to hemicolectomy because of recurrent attacks of diverticulitis and another because of other vulnerable diverticula of the part. Simple excision of the inflamed diverticulum with closure of the defect was performed in twenty-eight cases. Presumably these were instances in which the diagnosis was clear and the resected area was well removed from the ileocecal valve. The remaining eight cases were managed by a variety of rarely used procedures, namely, exploration followed by antibiotics, drainage of the area, inversion and purse-string suture or exteriorization of the segment.

There were no deaths in the hemicolectomy group, but wound sepsis was encountered in one case and phlebothrombosis in another. In the local resection and closure group three encountered major postoperative complications or prolonged periods of drainage and disability; likewise there were no deaths. Unfortunately long-term follow-up records were lacking. Of the fifteen patients reported as having "recovered" or "remained well," twelve were in the partial colectomy group which is of less value than had it been provided for the group not receiving colectomy. Of the twenty surgeons reporting these cases, thirteen indicated their preference for simple excision of the diverticulum with simple closure of the cecum whenever appropriate and practical. Hemicolectomy was the usual alternate procedure except for the very small lesion with

minimal or no regional inflammatory reaction which could be inverted. No one in this group indicated a desire to resort to the exteriorization procedure.

Symptoms. The symptomatology of cecal diverticulitis, which almost always comes to the attention of the physician in its acute phase, deserves only passing comment since about 90 per cent of the cases are diagnosed as acute appendicitis. Like the pathogenesis of acute appendicitis, the usual cause of an inflammatory process in a solitary diverticulum appears to be obstruction at the point of its communication with the gut. This is probably brought about by the presence of a fecalith with ulceration or by swelling and edema incident to some irritative lesion of the intestinal mucosa. In either situation a confined inflammatory process occurs which spreads through the lymphatics of the area rather rapidly with the production of further congestion, edema and induration. Gangrene and slough may occur. However, most often the area is sealed off quickly by the greater omentum and adjacent intestinal loops, confining the process locally as in appendicitis. If drainage occurs into the lumen of the gut, the process may quickly subside. If not, a paracecal abscess or chronic granuloma may be the result.

TABLE II

AUTHOR'S CASES

Year	Age	Sex	Case No.	Preoperative Diagnosis	Gross Appearance at Operation	Operation	Postoperative Course	Long-term Follow-up
1947	36	M	1	Acute appendicitis	Localized inflamed nodule, anterior wall	Appendectomy only; antibiotics	Satisfactory	Well 6 yr.; x-ray shows other diverticulitis of cecum
1950	47	M	2	Appendix out; right lower quadrant mass; ? carcinoma, barium enema inconclusive	Indurated cecal mass; ? carcinoma posterior wall	Right hemicolectomy	Satisfactory	Well after 3 yr.
1951	43	M	3	Tender right lower quadrant mass; ? appendiceal granuloma; ? carcinoma; barium enema inconclusive	Granulomatous mass; ? carcinoma posterior wall	Right hemicolectomy	Satisfactory	Well after 2 yr.
1951	23	F	4	Acute appendicitis	Inflamed nodule with fecalith, anterior wall near valve	Appendectomy only; antibiotics	Satisfactory	Well 2 yr.; follow-up x-ray shows deformed cecum
1952	51	F	5	Tender right lower quadrant mass; diverticulitis; diagnosis on barium enema	Localized inflamed mass anterior wall near valve	Cecectomy; ileocolostomy	Satisfactory	Well after 1 yr.
1953	38	M	6	Appendicitis; chronic recurrent	Localized inflamed mass anterior wall near valve	Right hemicolectomy	Satisfactory	Well after 1 yr.
1953	52	M	7	Acute appendicitis	Inflamed mass near valve with diffuse cellulitis	Right hemicolectomy	Satisfactory	Well after 1 yr.
1953	38	M	8	Appendix out; diverticulitis; confirmed by x-ray, recurrent right lower quadrant pain	Deformed cecum with fibrosis and induration	Right hemicolectomy	Satisfactory	Well after 1 yr.
1954	17	M	9	Acute appendicitis	Localized inflamed nodule, anterior wall	Diverticulectomy	Satisfactory	Well after 5 mo.
1954	26	M	10	Acute appendicitis	Localized inflamed nodule	Diverticulectomy	Satisfactory	Well after 5 mo.
1954	43	F	11	Acute appendicitis	Granulomatous mass posterior wall with regional cellulitis; ? carcinoma	Cecectomy and ileocolostomy	Satisfactory	Well after 6 mo.
1954	32	M	12	Chronic recurrent right lower quadrant pain and tenderness with deformity of cecum on x-ray	6 cm. mass on posterior wall; ? carcinoma	Cecectomy and ileocolostomy	Satisfactory	Well after 6 mo.

Differential Diagnosis. When the abdomen has been opened it is usually immediately apparent that one is dealing with a localized inflammatory lesion of the cecum, and since cancer of the cecum is the most frequent lesion encountered there, next to appendicitis, the problem of differentiation immediately becomes important in deciding upon the most definitive surgical attack with minimal risk to the

FIG. 1. Barium enema showing typical diverticula of the cecal pouch (incidental finding).

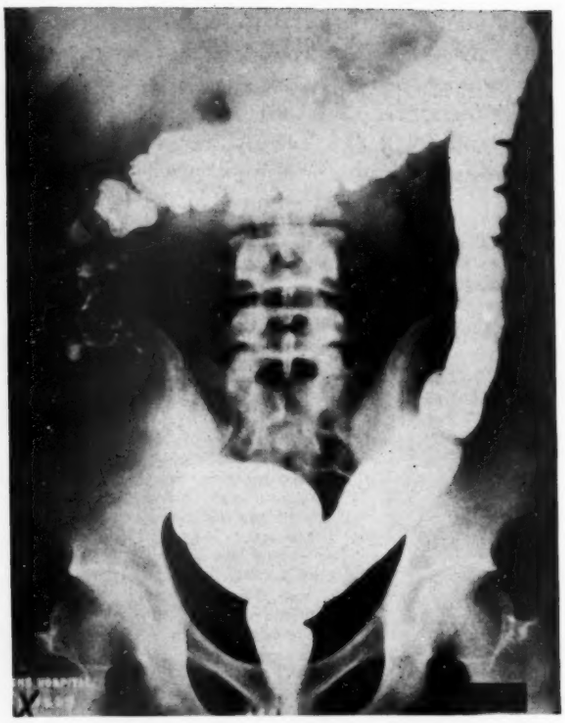

FIG. 2. Barium enema in Case VIII (author's series) showing marked irritability and deformity of cecal pouch due to frequent inflammatory episodes.

patient. The usual variety of carcinoma encountered in the cecum is the fungating type which projects for the most part into the lumen of the gut. However, the intramural variety which infiltrates the bowel wall, ulcerates and metastasizes rather early does occur.

Tuberculosis is the third most common condition, but usually involves the terminal ileum as well, and often small tubercles can be seen on the serosal coats. Usually there is considerable regional fibrosis and thickening of the gut wall with the presence of regional caseous nodes.

Actinomycosis must also be thought of, the most characteristic finding being the presence of multiple small abscesses, sinuses and fistulas which may liberate the classical sulfur granules.

Segmental diffuse ileocolitis of non-specific nature, like that of regional ileitis, has been encountered and it may be impossible to differentiate this condition from diverticulitis with diffuse lymphangitis and cellulitis of the cecal area.

Benign ulcers of the cecum have also been reported, a so-called non-specific variety, as well as those which may occur in amoebic infection. Although all of these conditions must be kept in mind, the essential and immediate problem is to exclude the presence of malignancy, if possible.

Comments. This disease continues to present a surgical problem of some diagnostic and therapeutic importance; the condition is probably not nearly so rare as once considered and it does occur at any age. The majority of patients are seen during the terminal period of the third decade of life when the incidence of appendicitis is decreasing and that of malignant disease is beginning to occur with greater frequency. There are no clinical manifestations or laboratory procedures, except barium radiography, which are of real assistance in making a preoperative diagnosis. (Figs. 1 and 2.)

Therefore, in the vast majority of instances a tentative diagnosis must be made at the operating table. The essential and immediate problem in diagnosis is the differentiation of an inflammatory lesion from a malignant one. Usually from reports in the literature and certainly from our own experiences this can be done. An inflamed diverticulum is usually on the surface and involves the external layers of the cecum. It is well localized and presents a surrounding area of induration and edema, and is likely to be adherent to a tongue of omentum.

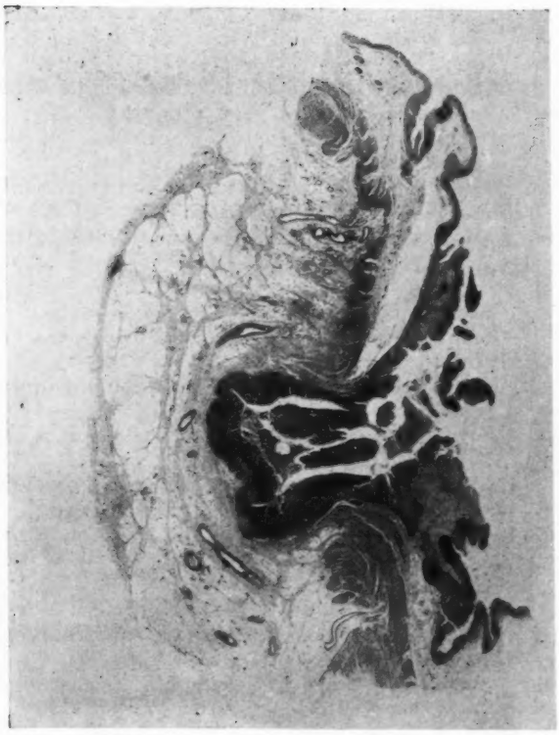

FIG. 3. Photomicrograph of chronic inflamed diverticulum with adjacent granuloma. (Case XII). Normal size.

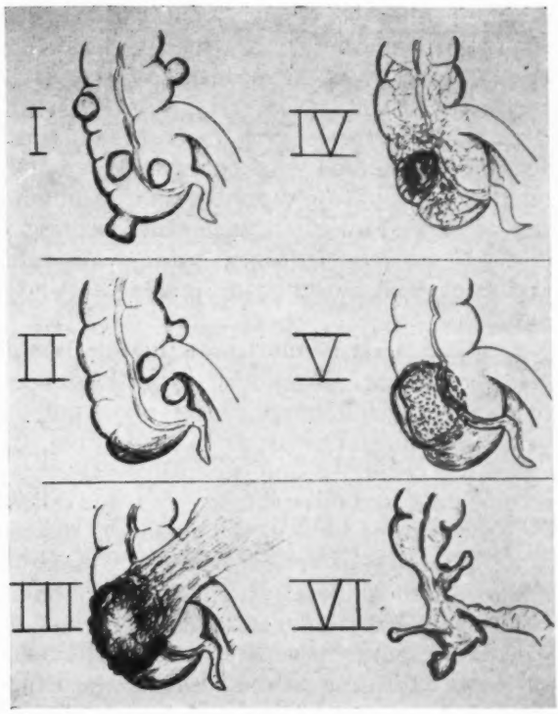

FIG. 4. Diagram showing several gross pathologic lesions encountered: I, diverticula of size and in locations suitable for local excision and closure; II, diverticula in location unsuitable for local incision; III, large inflammatory cecal mass with or without suppuration; IV, diverticulitis with diffuse intramural lymphangitis; V, posterior diverticulum with perforation and large adjacent granuloma; VI, deformed cecum from recurrent inflammation in diverticula with resulting fibrosis.

The most frequent sites are on the anterior and lateral aspects of the cecal pouch, but often are near the ileocecal valve. The classic, gross and pathologic characteristics of cecal cancer are well known and in the typical case there would be little question as to the diagnosis. However, carcinoma of the gut may at any time exhibit ulceration, induration and perforation with all other attendant signs of inflammation. Likewise, a primary inflammatory lesion due to fibroplastic reaction and granuloma formation may simulate malignancy. In certain cases the pathologist will be of great help with frozen section examination. (Fig. 3.)

Treatment. There is no substitute for the accurate evaluation of gross pathologic findings and the ability of the surgeon to render appropriate "on-the-spot" decisions in each case. From cases reviewed we cannot say that extensive resection at the time of discovery would be routinely desirable, since the case studies do not include information on long-term follow-up. If, following diverticulectomy and closure, simple drainage, or laparotomy and antibiotics alone, recurrences were recorded as common, complications were more severe or disability was greater, we might be justified in recommending routine right hemicolectomy as preferred treatment. Therefore, the selection of an appropriate surgical procedure must depend upon anatomic and gross pathologic characteristics of the lesion encountered in each case. (Fig. 4.)

From our experiences, supplemented with what we find in the literature, it would seem that certain generalizations regarding the proper treatment for these cecal lesions might be summarized as follows:

1. The discrete, well localized, inflammatory surface nodules seen on occasions should be treated by excision and simple closure provided (a) closure can be effected using normal cecal wall and without sacrifice of too great an area; (b) following turn-in closure, the normal function of the ileocecal valve would not be impaired; and (c) there should be no gross evi-

dence of other diverticula of the part. If these requirements cannot be met, we believe cecetomy or right hemicolectomy should be performed.

2. If a larger cecal or paracecal mass which suggests carcinoma is encountered, resection of the part in appropriate radical manner should be performed. If an abscess is present, drainage by extraperitoneal route is desirable and safer, with resection to be done at a later date.

3. More rarely a mural inflammatory nodule may be present, surrounded by rather extensive cellulitis and lymphangitis involving the entire cecum, terminal ileum and often the ascending colon. In our experience colectomy is to be preferred since the process is too diffuse for any type of local excision; and if treated conservatively, prolonged disability from a stormy febrile illness, which often results in intestinal obstruction, can be expected.

4. Those patients with an acute inflammatory right lower quadrant lesion whose appendices have been removed should be treated conservatively until diagnostic studies are done. If carcinoma can be ruled out, the decision regarding resection should depend upon the frequency of attacks, disability and cecal deformity produced.

5. At the present time there is no evidence that uncomplicated, asymptomatic diverticula of the cecum discovered in the course of barium enema should receive surgical treatment of any type.

6. When resection of an unprepared colon is undertaken the principles of using neomycin solution at the operating table, as described by Poth,[5] should be remembered.

Acknowledgment: Appreciation is extended to Drs. Burgess, Freeman and Cherry for permitting use of cases treated by them.

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DISCUSSION

C. E. Hardwick (Portland, Ore.): I would like to congratulate Dr. Waite on his fine paper. After reading his manuscript my first reaction was similar to the surgeon who, on opening an abdomen thought to contain an acute appendix, was confronted with an inflammatory process yet undiagnosed. There is bound to be a certain amount of frustration associated with such a discovery.

Fortunately the proctologic surgeon is not often presented with such a problem. The abdomen presenting signs of acute appendicitis is most frequently seen by the general practitioner or general surgeon. However, this situation may more commonly confront one of us on being called into surgery to aid in differential diagnosis of cancer of the cecum from an inflammatory process.

Often in retrospect our taillights turn much

brighter than our headlights. If we had seen the patient before the abdomen was opened and had taken the opportunity to obtain a detailed history, the astute surgeon might obtain even negative information that might be helpful in determining the course of action to be taken at surgery. Too often we fail to obtain pertinent facts preoperatively in apparently simple acute appendicitis. The following questions should be asked: (1) Has this patient had an unexplained anemia? (2) Has there been any visible blood loss? (3) Has the patient ever palpated a mass in the abdomen? (4) Are there any symptoms suggesting obstruction? (5) What is the patient's general systemic condition?

We must realize that many who read Dr. Waite's paper on publication will represent the occasional operator who may be faced with this momentous decision. He must have in his armamentarium the knowledge of the simplest and safest expedient. I am fully in accord with the suggested methods Dr. Waite has summarized. I would not discard exteriorization as an available solution to the problem.

Because this unexpected development does occur when the surgeon least expects it and if differential diagnosis of carcinoma of the cecum or some lesion amendable to right colectomy was decided upon, consideration at that point must be given to: (1) the skill of the surgeon; (2) the general condition of the patient for more extensive surgery; (3) the type of anesthesia permitting extending the field of surgery; (4) the availability of blood; (5) the availability of antibiotics such as aureomycin; (6) the presence or absence of obstruction.

A higher mortality and prolonged morbidity are to be expected if the type of lesion is mismanaged. I was recently asked to manage a diverticulitis of the cecum six weeks after the original appendectomy and cecostomy had been performed. Unfortunately this procedure, done by a general surgeon, had not been adequate because of the obstruction not noted at the time of surgery. Multiple small bowel fistulas through McBurney's incision ensued, resulting in marked fluid and electrolyte loss and skin excoriation. A right colectomy together with resection of considerable terminal ileum resulted in a cure.

The Permanent Cure of Hemorrhoids

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The percentage of five-year cures for neoplastic disease of the colon, rectum and anus has shown consistent improvement as revealed by numerous statistical studies. By contrast, the percentage of five-year cures for hemorrhoids has shown little change in the last half century. Better hospital facilities and anesthesia have been responsible for any improvement that has occurred.

The purpose of this paper is to present a surgical approach to hemorrhoidal disease more radical than any previously described. It does not agree with the oft-stated premise that the best operation is the one with which the individual surgeon is most familiar. It does not accept injection treatment as curative of hemorrhoidal disease. Rather, it is based on a definite concept of the pathology of this condition and that the disease is readily accessible, lending itself to complete removal and cure in all cases. This paper subscribes to the surgical principle that cure of any condition depends on the complete excision of all disease.

The histories of 1,600 new patients seen from July, 1948 to July, 1953 were examined to determine what percentage of those needing hemorrhoidectomy had had previous surgical or injection treatment. Any patient having a fistula or an ulcer who had previous hemorrhoidectomy was included.

A total of 898 patients were advised to have surgery; 554 underwent operation. Of these, ninety-four or 17 per cent were having a second hemorrhoidectomy. It is interesting that ninety-three (same percentage) had one or more series of injection treatments. Forty-five or 8.1 per cent had both injection treatment and previous hemorrhoidectomy. Fourteen or 2.5 per cent were having their third operation for hemorrhoids.

Seventy-eight patients or 4.9 per cent of the total number were given injection treatments. Of these, thirty-nine were injected because they refused surgery.

Of the ninety-four patients who were returned for a second operation, none of them requested it. All asked if there was not some other treatment.

Of the ninety-three patients operated upon after previous injection treatment, fifty-four requested further injection treatment. The remaining thirty-nine preferred surgery.

A colleague reported to me that 19 per cent of his last 200 hemorrhoidectomy patients had had previous anal surgery and 28 per cent had had previous injection treatment.

Fansler[1] reported that 24 per cent of his patients had previous anal surgery. Buie[2] reported 20 per cent of 566 surgical patients had been operated upon previously.

There are two principle factors responsible for the high percentage of patients requiring a repetition of their anal surgery: First, there is no uniform interpretation of the pathology. I agree with older writers[3,4] who, for the most part, classified internal hemorrhoids as venous, columnar and nevoid. In the venous type, large varicosed radicles predominate the findings. In the columnar, a redundant hypertrophic rectal mucosa predominates and covers the venous enlargement. In the nevoid, a more red, easily bleeding pile containing an obviously greater arterial component is seen.

Tucker[5] contends that increased arterial flow and arteriovenous connections are important factors in the production of hemorrhoids. Some investigators[6-8] have reported an increased number and size of arterioles within different hemorrhoidal masses. No direct evidence of arteriovenous connection or changes in arterial walls has been demonstrated.

It must be brought out here that it is not necessary to have changes in the wall of an artery for it to carry increased blood to a part. When arteriovenous connection occurs in smaller vessels, it is only the pressure in the venous side that is changed and only changes in the wall of the vein result.

It is illuminating to consider, for a moment, some concepts of pathology in other parts of the body.

Wakim and Mann[9] have demonstrated

arteriovenous connections between the hepatic artery and portal vein within the liver. McIndoe,[10] using this information plus his own extensive studies, has demonstrated that portal pressure is actually determined by the inflow of blood through the hepatic artery. This has been the basis of ligation of the hepatic artery to reduce portal pressure in cirrhosis of the liver, as reported by Reinhoff.[11]

Pratt[12] has demonstrated the existence of minute arteriovenous connections in 16 per cent of people with varicose veins. These connections are multiple and must be interrupted to obtain a surgical cure of this disease. Trauma, infection and intimate arteriovenous relationship have long been recognized as the factors responsible for establishing a connection between the two systems. All this certainly occurs in the anorectum.

Hemorrhoids must be considered to be a basic vascular disease consisting not only of masses of dilated veins but also, frequently, arteriovenous tumors. The mucosal and skin coverings always share in the process. Although localized areas of greater venous dilation do occur, the veins involved encircle the lower rectum and anus and there is no interruption of continuity in their arrangement.

All patients having hemorrhoidal disease have dilated venous radicles extending around the anal canal. There has also been an extension of pressure longitudinally so that some degree of internal and external hemorrhoids is always present no matter which area was primarily involved.

At autopsy I dissected the rectum and surrounding areas of two infants, one of each sex. The veins of the vesical, prostatic and superior hemorrhoidal plexuses were dissectable in the male, and the superior hemorrhoidal and vesicovaginal plexus in the female.

The veins of the superior hemorrhoidal plexus surrounded the area evenly in both. In no area were the veins more numerous or larger than any other. The external hemorrhoidal plexus could not be recognized as such at birth.

Any localized areas of venous distention as found in hemorrhoidal disease seem not to be present at birth. Clinical experience leads me to believe that the location of these areas is determined more by the location and degree of arterial supply than by the location of any large veins which drain the area. I do not believe it is significant that the superior hemorrhoidal vein is part of the portal system.

If this concept of the pathology of hemorrhoids is accepted, it becomes apparent that to cure them permanently a procedure must be followed which, first, recognizes and removes any excess rectal lining. Second, it must not only excise the presently dilated veins but also remove the veins of both plexuses which might in the future dilate to form more hemorrhoids. Third, the procedure must interrupt the arterial flow which is at the time and which may later contribute to more venous dilation. Fourth, it must serve to correct other coexisting anal pathologic disease. Last, it must provide a smooth functional anal canal and perianal skin.

The second factor responsible for the return of hemorrhoids after surgery is a fear which prevails that an operation radical enough to accomplish the aforementioned will be followed by frequent shortening of the anal canal or anal stenosis and a prolonged period of morbidity.

The operation I perform routinely is in many respects like that described by Fansler and Anderson[13] in 1933. They recommended a plastic type operation for "large, prolapsed, thrombosed hemorrhoids surrounding the entire anal ring." However, they use a less radical procedure for other cases.

The procedure described by Granet[14] recently was a plastic procedure to prevent recurrence. Other men[15,16] have used this type of operation on selected cases.

TECHNIC

A Fansler operating anoscope is used because it provides adequate exposure while maintaining normal anatomic relationship. After determining the size of the anus under anesthesia, the anoscope is inserted with the window to the patient's right. A large Allis forceps grasps the tissue at the anal verge. A suture of No. 00 chromic catgut on a small round needle is next placed through the rectal mucosa above the upper reaches of the internal hemorrhoidal tissue. Traction is then made on this suture to test the redundancy and mobility of the rectal lining. The upper point that is easily brought through the scope to the anal verge determines the upper level of mucosal excision. Excision is then made of the tissue included in the large Allis forceps. This is

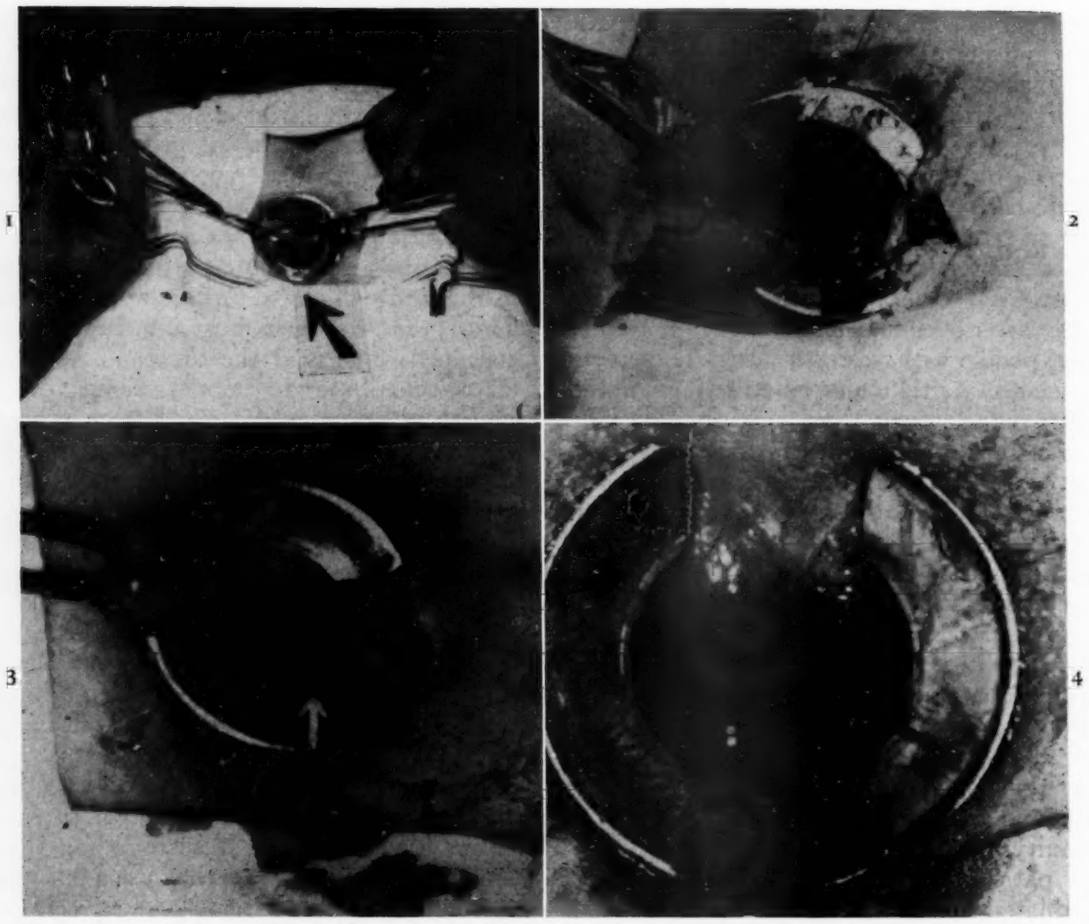

FIG. 1. Arrow indicates suture; shows height of excision. FIG. 2. A, sphincter border; B, internal sphincter. FIG. 3. Arrow indicates sutured mucosa. FIG. 4. Arrow indicates residual hemorrhoid posteriorly; two lateral areas have been removed.

extended upward including a strip of anal skin and a larger triangular section of rectal mucosa to the previously placed suture. The mucosal defect thus created is sutured longitudinally to the level of the intersphincteric line. This has seemed, to me, to effect lengthening of the anal canal. (Figs. 1 to 3.)

The scope is now rotated 180 degrees and the procedure repeated on the left side. This results in two bilateral mucosal defects, each closed longitudinally. Most of the internal hemorrhoidal tissue has been removed as well as all excess rectal mucosa.

The window of the instrument is then placed posteriorly. A suture is now placed just above the remaining hemorrhoids in this area. Next, a transverse incision is made connecting the posterior edges of the bilateral anal wounds. A wide flap of posterior anal skin is now dissected free. If an ulcer is present, it does not alter the technic. All external hemorrhoidal tissue is excised from the surface of the external sphincter out beyond the border of the superficial portion. Any adherent hemorrhoidal tissue is removed from under the surface of the skin flap. (Fig. 4.)

A posterior sphincterotomy is now performed as a routine procedure. The degree is determined by the size and condition of the anal canal. (Fig. 5.)

Years ago I did not perform sphincterotomy routinely but experience has taught me its value. Nothing will reduce postoperative pain as much as cutting the sphincter. While it prevents postoperative stenosis, it does not produce incontinence.

Next, the remaining cuff of internal hemorrhoidal tissue is removed. The skin flap which is wide and large is sutured to the rectal mucosa transversely with atraumatic No. 00

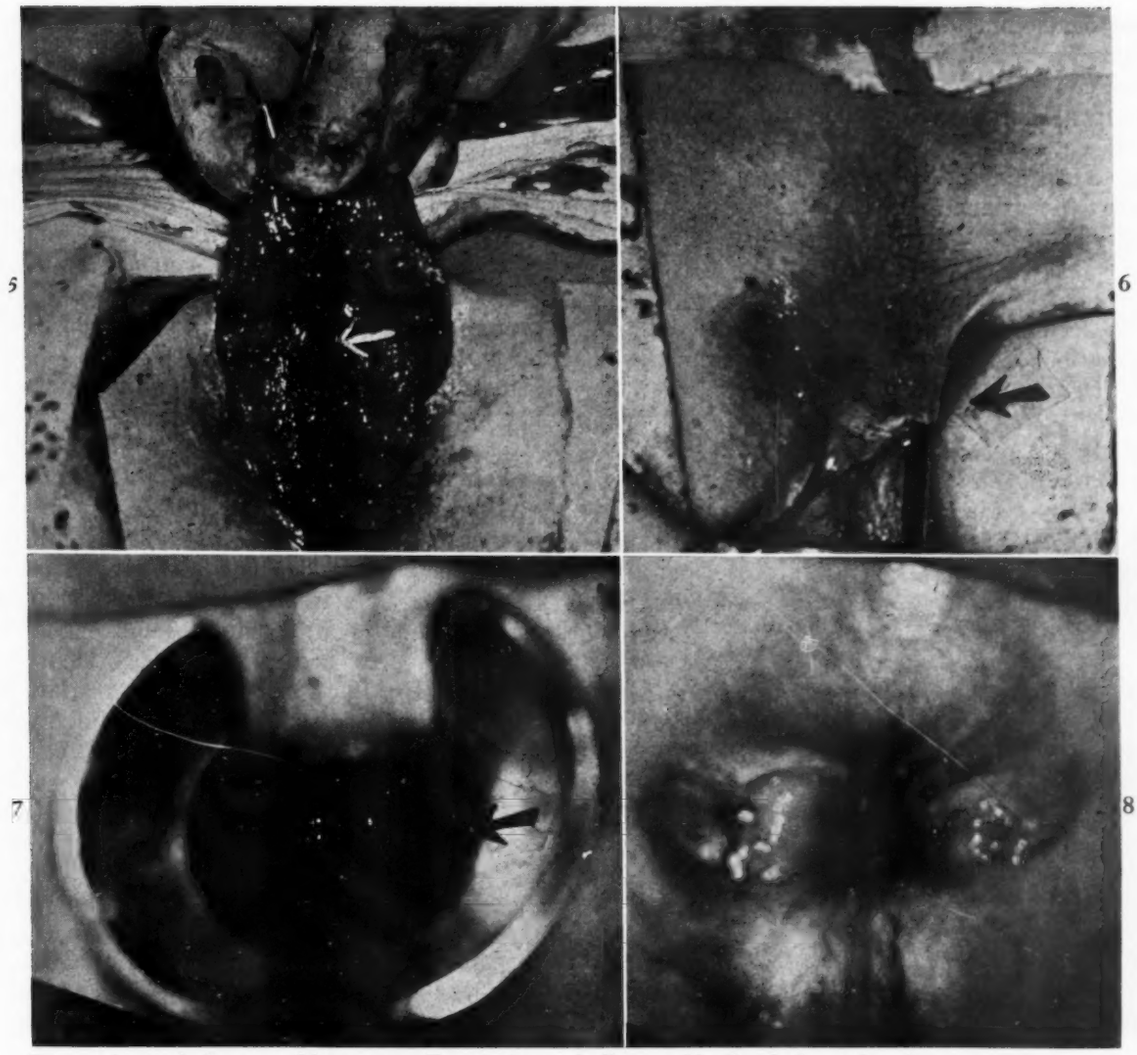

FIG. 5. Arrow indicates sphincter cut. FIG. 6. Arrow indicates skin flap before trimming, resuture or reattachment to rectal mucosa posteriorly. FIG. 7. Arrow indicates skin flap replaced by suturing. Flap replaced over defect posteriorly and over area of sphincterotomy. FIG. 8. Completed operation.

chromic suture. After this, the same procedure is performed in front except that the sphincter is not cut. Now, all internal and external hemorrhoidal veins have been removed from the exposed surface of the entire anorectum. The arterial supply has been divided. The return of hemorrhoids is prevented by removing all pathologic components. (Figs. 6 to 8.)

COMMENTS

Seven hundred ninety-two operations of this type have been performed. The first of these were less radical than the present procedure.

The average hospital stay is five and a half days postoperatively. The average time away from work is twelve days. Thirty per cent have some postoperative narrowing, most pronounced at twenty-five days but which has softened and disappeared by ninety days. One per cent complain of narrowing for six to eight months. Only seven have had any narrowing to examination at the end of one year. A total of five patients have returned to surgery for further proctotomy and residual posterior ulcer.

No patient has had incontinence or recurrence of hemorrhoids, and in no patient has a fistula developed.

The advantages over other procedures of a plastic type as described are: (1) All excess rectal mucosa is removed. This produces a good functional anorectum and tends to prevent

the development of rectal prolapse. (2) All anal crypts and papillae are excised. (3) The anal canal is not shortened. (4) Anal narrowing is at a minimum. (5) Posterior proctotomy with suturing of a large postanal skin flap to the rectal mucosa has seemed an ideal way to cure existing anal ulceration and to prevent it.

SUMMARY

1. Evidence is furnished showing need for an operation which will reduce or eliminate a recurrence of hemorrhoids.

2. A concept of the pathology of hemorrhoids is advanced and evidence presented to support this concept.

3. A surgical procedure has been developed which excises the entire pathologic process.

4. The operation does not increase complications or morbidity and effects a permanent cure of the disease.

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DISCUSSION

Albert G. Schutte (Milwaukee, Wis.): Undoubtedly there is much to be desired and strived for insofar as improvement of the operative results of hemorrhoids is concerned. However, the fact that a fairly large number of patients require repeat hemorrhoidectomy does not necessarily mean that certain well established and properly executed methods of hemorrhoidectomy are at fault. It does often mean that the operator lacked the experience, judgment and operative skill necessary to produce a good result. It could also mean that inadequate postoperative care was at fault.

It is certain that the operative cases which have been done by the members of this organization show a far smaller incidence of the need for repeat hemorrhoidectomy than was found by Dr. Parkinson. It is certain that the surgical results of any single surgeon of this organization improved as he modified and perfected his surgical technic and acquired greater experience and judgment in the practice of proctology. As the influence of this Society has grown during the past fifty-four years, it is also certain that the efforts of its members, through their private practice and their teaching, have done much to improve the over-all end results of hemorrhoidal surgery.

Patients needing hemorrhoidectomy present a wide assortment of gross pathologic pictures which are the result of varied anatomic changes that have taken place in the lower rectum, anorectal line, anal canal, anal area and anal sphincters. The gross pathologic changes are relative and tend to be progressive. The twenty-five year old patient with marked pathologic changes offers a poorer prognosis for permanency of results than does the forty-five year old patient with similar abnormalities. In other words, the potential for developing new abnormalities after operation appears greater in the young adult.

Clinically, it is apparent that some hemorrhoidal cases show an unusually abundant arterial supply. This rich arterial supply appears significant as an etiologic factor and must be dealt with, and usually is dealt with, in the surgical management of the disease.

Dr. Parkinson lists five essentials for an

operative procedure designed to cure hemorrhoids permanently. This list, based upon the concept of the pathology outlined by him, is very inclusive. The accomplishment of these essentials by operation undoubtedly would produce an excellent result. However, inasmuch as the inherent predisposition to hemorrhoidal disease as well as the type and degree of abnormalities are variable in different individuals and because there may be a multiplicity of contributing causes, the problem of recognizing and removing all of the veins which might in the future dilate to form more hemorrhoids is a perplexing one, as is also the problem of recognizing all of the arterial flow which might later contribute to venous dilation.

Dr. Parkinson is right when he states that one of the factors responsible for the need of repeat hemorrhoidectomy is the fear that operations radical enough to produce permanently good results are likely to cause complications or prolonged periods of morbidity. It appears, however, that the inadequacy of the surgeon would largely be the cause for the alleged fear of the untoward results of radical methods of treatment. The radicalization of any hemorrhoid operation must not only be commensurate with the anatomic abnormalities present but also, when possible, should include the potential sources of abnormalities.

Dr. Parkinson's operation is extensive and inclusive; and if surgeons learn to perform it as well as he does, the permanency of the results attained should be a credit to the profession. It is by the efforts so well displayed in Dr. Parkinson's essay that progress in proctology is made.

ALTON OCHSNER (New Orleans, La.): I think Dr. Parkinson has shown that hemorrhoidal surgery is badly done generally throughout the United States and that there is need for change as he has demonstrated it. I wish that I could agree with him as regards his philosophy of the cause of hemorrhoidal disease, and I rise not as a proctologist, because I am not a proctologist, but a general surgeon who is interested in vascular diseases among others.

His theory that hemorrhoidal disease is primarily arteriovenous is a provocative one, but I cannot agree with him that most hemorrhoids are the result of arteriovenous fistula. Dr. Parkinson and I discussed this and I hate to disagree with him because he and I have been good friends for a long time.

There is only one way in which one can determine this and that is to determine the oxygen concentration of the blood in the hemorrhoidal veil and compare it with the venous blood in other parts of the body. He may be correct, I do not believe he is, but it can be determined once and for all by getting oxygen saturation studies on the blood of hemorrhoidal veins and in that way it can be studied.

E. D. PARKINSON (closing): I must say that I agree 100 per cent with what Dr. Schutte has said about the results of the improvements in anal surgery as done by members of this Society. There can be no question about that. Regarding Dr. Ochsner's talk, he of course is in a better position than I am to discuss etiology of any primary vascular disease. I did not mean to say that *most* hemorrhoids are due to arteriovenous connections. Hemorrhoids are primarily a vascular disease in which the venous enlargements are produced by a retrograde venous pressure much the same as varicose veins elsewhere are produced. Arteriovenous connections do occur and contribute to the increased venous pressure in a certain percentage.

I did hope someone would take up my point that I do not believe it is significant in the etiology of hemorrhoids that the inferior mesenteric vein is a part of the portal system.

I have never seen varicosities in the mesosigmoid. Varicosities of the broad ligament are common.

Redundancies of the Rectal Mucous Membranes

THOMAS WILENSKY, M.D., *Lansing, Michigan*

VOLUMETRIC excesses of rectal mucosal lining disposed in the circumferential as well as in the longitudinal distribution are productive of symptoms infrequently attributed to them. This is particularly true in the presurgical patient whose recognizable anorectal disease is held accountable for all complaints. In the postsurgical anorectal patient failure to have dealt with the abnormal mucosal folds is frequently responsible for the persistence or modification of the original complaints.

In the scant available literature pertaining to this subject a persistent confusion of nomenclature and definition involving hemorrhoids, mucosal redundancies, prolapse and procidentia has been found.

Rectal prolapse of the mucosal variety is by definition not distinguishable from the abnormality considered herein. Neither procidentia, meaning descent of all coats of the rectal wall, nor high internal rectal prolapse, the so-called sigmoidorectal intussusception, is included in this paper.

While they merge into whatever internal hemorrhoids are present, symptom-provoking rectal mucosal redundancies are not to be regarded as particularly high internal hemorrhoids as the point of division is readily apparent to those who have familiarized themselves with this region. Oftentimes, particularly in youthful patients, the actual hemorrhoidal development in the presence of large overlying rectal mucosal folds is insignificant.

This observation is offered in support of the premise that volumetrically excessive rectal mucosal membranes constitute a true congenital disturbance with a strong familial predisposition. Similarly, it tends to refute the statement that large prolapsing rectal mucosal folds in the presence of large mixed hemorrhoids are secondary to the hemorrhoids. On the contrary it suggests strongly the secondary nature of the hemorrhoidal developments.

The rectal mucosal folds constituting the focal points of this study lie in the extreme distal 2½ to 3 inches (6½ to 7¾ cm.) of the rectum. This arbitrary measurement was selected following the observation that this was the extent to which surgical extirpative measures could be applied with safety and satisfactory long-term results.

Clinical Significance. Redundant rectal mucosal membranes occurring alone, in association with significant anorectal disease, or observed in the postoperative anorectal patient are likely to produce symptoms which lack specificity and which require careful and experienced interpretation. Frequently patients in whom this condition has been overlooked during other anorectal surgery or in whom anorectal surgery of any type was not advised have been classified as psychoneurotics.

Many years ago Alvarez[1] dispelled the popular notions of toxic absorption from the colon. He produced similar symptoms by packing the ampulla with cotton. This lends support to the speculation that large, sagging, congested, ampulla-filling and obstructing rectal mucosal folds interfere with the regional autonomic nervous impulses giving rise to such systemic complaints as headache, backache, legache, feelings of fullness, dragging sensations in the lower abdominal quadrants and feelings of lassitude and fatigue. Rectal fullness only temporarily relieved by defecation and intensified as the day wears on has been a commonly encountered complaint.

Frequent unsuccessful defecatory efforts are likely to be interpreted as constipation even though bowel movements occur daily. Resort to laxatives and enemas usually aggravates the condition. The nagging rectal sensation disappears during sleep and frequently over weekends of rest and relaxation.

A chronically moist itching anus is a common presenting complaint, particularly in postoperative anorectal patients in whom the mucosal abnormality has been unrecognized and hence

surgically ignored. Rectal mucosal folds overlying the internal orifice of the anal canal and even dilating and entering into the canal with the seepage of mucus, sometimes stained with feces, is the probable cause of this symptom. In such pruritic cases the perianal skin, in my experience, has rarely borne the characteristic appearance of the usual variety of pruritus ani. For the most part, these patients are troubled during the day and are almost invariably symptom-free at night and during week-ends and vacations.

Flatulence has been a third prominent complaint. At the present time I can only suggest that this is more than likely a manifestation of autonomic interference. However, its common occurrence, relieved by excision of the mucosal redundancies, has been observed often enough to convince me that such a relationship does exist.

Sacral backache and/or coccygodynia with or without sciatic radiation is an additional complaint which has responded to treatment directed to the rectal mucosal redundancies. Several patients, whose posthemorrhoidectomy complaints of perineal uneasiness and a tendency to dysuria and frequency had led to unsuccessful treatment of questionable urinary tract infection and prostatitis, were relieved when the rectal mucosal abnormality was recognized and terminated.

Diagnosis. The mucosal folds under consideration are normal except for their excessive volume in both the transverse and longitudinal directions. Although hypertrophic proctitis is sharply limited to the distal several inches of the rectum it is, until proved otherwise, a manifestation of chronic ulcerative colitis and has no part in this discussion.

Inasmuch as there is no substitute for the meticulous visual scrutiny of the terminal bowel, without which elective anorectal surgery cannot be justified, it appears obvious that the redundant mucosal folds must be identified by this means.

The manner in which the examination is conducted is of considerable importance in order that extensive and symptom-causing mucosal folds are not regarded clinically insignificant during a rapid routine proctosigmoidoscopic examination. The preparation of the patient with enemas, his relative inactivity for some time prior to examination and the recumbent position will contribute to some actual decrease in size of the presenting folds. Add to this the massaging effect of the preliminary digital examination followed by the mucosal reduction produced by the advancing hollow instrument and the effects of gravity in the inverted patient, and it becomes apparent that the overlooking of large mucosal folds is not unlikely.

Because the patient's complaints are most prominent when he is in the upright position and particularly when intra-abdominal pressure is increased it seems advantageous and natural to reproduce such conditions for more accurate appraisal of the ampullary lining. This may be accomplished by asking the patient to strain forcibly with and without the short speculum in place. On occasion it is of great value to observe the patient while he is straining in the lithotomy or squatting positions.

Comprehensive pelvic examinations in women are essential in order to recognize visceral descensus with or without enterocele as important contributing factors to the rectal disability. Prostatism and seminal vesiculitis must be considered in men. Frequently one or both conditions are associated, clinically significant disclosures which may indicate a prudent delay in the application of surgical treatment to the anorectum until the relative importance of the separate conditions can be more critically assessed.

Treatment. Personal experience has convinced me that lasting relief is to be obtained only by surgical ablation of the offending mucosal folds.

Obviously this is best accomplished during the course of a single operation performed most commonly for the removal of mixed hemorrhoids. However, I have not hesitated to do so during more extensive anorectal procedures.

Two small series of patients were treated for this condition by sclerotherapy using 5 per cent buffered aqueous quinine and urea hydrochloride. Included were a few of my own relatively recent preoperative patients and a few personal postoperative patients in whom operation had been performed some years ago before my presently held concepts had taken shape. A larger number of patients operated upon elsewhere completed this group. Sclerotherapy was temporarily helpful in a number of patients. Liberal quantities of the agent were employed and injections were delivered to multiple foci at each treatment. Subjective relief lasted only during the periods of injections

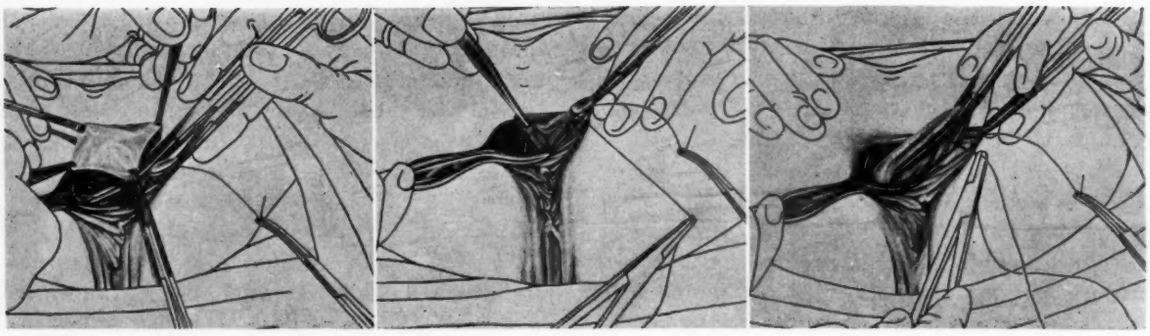

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FIG. 1. The diamond-shaped portion of excised mucous membrane, submucosa, muscularis mucosa and even some smooth muscle fibers.

FIG. 2. The hemostatic ligature has been applied on the mucous membrane, the redundancy of which is being tested preparatory to the application of the crushing clamp.

FIG. 3. The clamp has been applied widely and deeply and is undersewed back and forth with the original No. 00 chromic catgut atraumatic suture.

and for a subsequent time while the submucosal inflammatory process was still in its exudative stage securing some fixation of the loose dependent folds.

Because of my dissatisfaction with this method of treatment its use has been discontinued except in minor cases or when a patient is particularly insistent to see what could be accomplished before resorting once more to dreaded rectal surgery.

When one undertakes to correct this condition surgically the most important fact to keep in mind is that the excessive mucosal lining is disposed in a transverse or annular fashion, on occasion as much or even more than in the longitudinal direction. Failure to recognize this easily demonstrable situation accounts for the common employment of surgical methods which cannot correct the entire problem.

Amputation technics which have no reducing effect on the excessive circumferential expanse of mucous membrane will frequently fail to relieve the symptoms. Although the mucosal redundancies are generalized throughout the rectal ampulla they can, for purposes of excision, be gathered into the right posterior, right anterior and left lateral zones for combined removal with the major mixed hemorrhoidal formations.

When these sectors are adequately resected the intervening laxities are usually so well taken up as to require no additional attention. When such an added step is infrequently indicated I have employed Goodsall's technic transversely in the posterior midline well above the pectinate line using a T-clamp to facilitate the application of the ligature.

The circumferential reduction of the excessive mucosal lining has been accomplished by the longitudinal application of the crushing clamp deeply and to a high level. More than mucous membrane is included, particularly in the right posterior quadrant which is usually treated first and into which sector the largest mucosal redundancy is most readily gathered. Histologic study of the tissue removed demonstrated rectal mucosa, submucosa, muscularis mucosa and some smooth muscle wall. It seems reasonable to believe that the repair of such a defect will produce a more enduring effect than an excision limited to the mucosa alone. The sections removed have been diamond-shaped with the widest level well within the ampulla, rendering the possibility of stenosis or stricture most unlikely. (Fig. 1.)

The method of suturing has been considered carefully because the deep high application of the clamp encroaches upon blood vessels from which severe hemorrhage difficult to control could result. In addition, the thickness of rectal wall excised removes the barrier between the rectal lumen and the perirectal spaces in which an infective process could conceivably develop to serious proportions.

Technic of Suturing. A hemostatic suture ligature of atraumatic No. 00 chromic catgut is placed on the mucosa immediately above the pectinate line in the right posterior anorectal quadrant. (Fig. 2.) The needle is passed through the tissues several times in order to

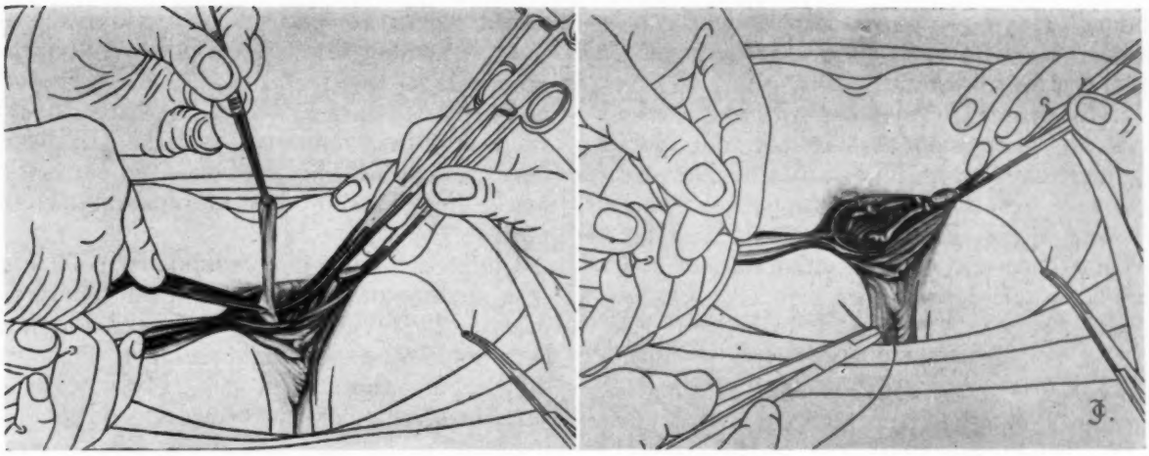

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FIG. 4. The undersewing completed, the redundant rectal mucous membrane is excised over the clamp which is then removed.

FIG. 5. The original suture is returned to the starting point in the crushed mucosal edge as a running interlocking hemostatic stitch and tied.

prevent slippage. The suture is now held tautly away in a radial direction while the overlying rectal mucous membrane is tested for mobility and extent. With the mucosal fold tented up in the rectal ampulla as high as seems indicated a Buie crushing hemorrhoidal clamp is deeply applied from a point immediately above the ligature. (Fig. 3.) The clamp is then undersewed back and forth with the originally placed suture until the tip of the clamp is reached. At this point the suture is locked, the tissue above the clamp excised and the clamp removed. (Fig. 4.) The suture is now returned in the crushed mucosal edge as a running interlocking hemostatic stitch to the starting point where it is tied. (Fig. 5.)

The mixed hemorrhoidal formation is now excised down to muscle through an elliptic skin incision. The anoderm is approximated with a running interlocking No. 000 chromic catgut suture beginning at the point of termination of the mucosal suture. An identical but commonly less extensive procedure is performed in the right anterior and left lateral sectors. Quite often the extensive right posterior mucosal excision alone almost achieves the objectives of the procedure.

Whenever pectenosis, sphincteric fibrosis and shortening, postoperative stenosis or stricture and anal ulcer or fissure are found, these conditions are dealt with through a radial incision in the left posterior anal quadrant and pectenotomy and/or partial sphincterotomy and regional hemorrhoidectomy are carried out.

Quite uncommonly mucosal excision as a separate step is found necessary in the posterior ampullary midline. This is accomplished by introducing Goodsall's ligature of chromic catgut, No. 1, on two or three needles, through the transversely disposed mucosal fold held in a long T-clamp. When the needles are cut out the ligatures are tied, securing the redundancy in three or four equal portions, the caps of which are excised with scissors.

SUMMARY

1. Redundant epithelial folds in the rectum and anal canal represent a common congenital disorder which is infrequently recognized as a clinically important and surgically significant cause of anorectal and systemic complaints.

2. Such mucosal redundancies are not merely the normal lining which has slipped to a lower station but are actual volumetric excesses which are disposed in the circumferential as well as in the longitudinal distribution.

3. Surgical disregard of these folds during other indicated anorectal surgery classifies the entire procedure as incomplete.

4. It is not possible to rectify this defect by amputating a portion from the end of the mucosal tube without directing deliberately planned surgical correction to the circumferential or transverse oversupply.

5. A modified safe and hemostatically secure surgical technic for radical ablation of the redundant rectal mucosal folds is described.

6. This technic, for which no originality is

claimed, may be employed independently or in association with the surgical treatment of other anorectal disorders.

7. In a limited experience with this method it is my impression that it has contributed significantly to improved early and late postoperative results. Complications have been few and minor. Early or delayed hemorrhage has not been encountered; stenosis and stricture have not occurred.

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DISCUSSION

Arthur H. Wells (Cincinnati, Ohio): As Dr. Wilensky stated in his paper, there is very little literature available on the subject of redundant rectal mucosal folds. My own personal experience is limited also. I believe that this subject is very closely allied to that of incomplete rectal prolapse and it may even be the same condition, depending upon one's interpretation of the findings during examination.

Dr. Wilensky mentions the relative frequency of redundant rectal mucosal folds in infants and children and I have found this to be true. However, I had always believed the condition to be due to the greater relative length of the rectum and colon in the infant and child as compared to the adult, and that as the pelvis changes in size, the condition tends to correct itself.

In discussing the primary or independently occurring redundant rectal mucosal folds the symptoms mentioned are: rectal fullness, chronically moist itching anus, flatulence, sacral backache and/or coccygodynia. We must remember that other conditions above or adjacent to the rectal region may produce any one or all of these symptoms, and although there may be a redundancy of the rectal mucosa, these conditions must be ruled out before attributing the symptoms to redundant rectal mucosal folds.

Under the subject of redundant rectal mucosal folds associated with significant anorectal disease, Dr. Wilensky has stressed the fact that not only is thorough examination necessary but also thorough surgery. I believe that a third factor should also be mentioned; surgical judgment. This can be gained only through experience.

Under the heading of Diagnosis Dr. Wilensky emphasizes the examining position. He believes that the examination should be performed with the patient in the position most likely to reproduce the condition. It has been my experience, in so far as I can recall, that most patients in such a position tend to fill the rectum with mucosal folds when intra-abdominal pressure is increased. I wonder if the presence of redundant rectal mucosal folds, while in an examining position less likely to demonstrate them, might not be of greater symptomatic and pathologic significance.

In treating the condition with sclerotherapy temporary relief has been obtained. I believe that if this method of treatment were always tried before surgery was performed, a few unnecessary operations might be prevented in cases of primary or independently occurring redundant rectal mucosal folds. In other words, it should be a good therapeutic test for determining whether the redundant folds are symptomatic. As Dr. Wilensky has pointed out in his discussion of surgical technic, it is important to remember that there is an increase in the amount of rectal tissue in the circumferential as well as in the longitudinal direction, and excision in one direction alone could not bring about the desired reduction in the amount of tissue present.

I believe that many of us have gained a new concept of redundant rectal mucosal folds from Dr. Wilensky's comments.

Anal Sphincter-preserving Operation for Congenital Low Rectovaginal or Rectoperineal Fistula

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THE objective of plastic correction of imperforate anus is to obtain a surgical anus that will function as nearly to normal as possible. Since 1857, Rizzoli's[1] operation has been the procedure of choice of most surgeons for correcting congenital imperforate anus with associated low rectovaginal fistula. In 1936 Stone[2] described a procedure which he employed with excellent results in three patients with this anomaly. The advantages of his operation over that of Rizzoli are that the perineal body and anal sphincter muscles are not severed and the mobilized fistulous opening with the lower rectum are transplanted to the normal anal location. Despite these advantages the procedure has not enjoyed wide popularity. In a cursory review of the literature since Stone's report, only four additional cases were found of patients whose anomaly was repaired in this way.[3–5] However, there are undoubtedly a number of unrecorded cases in which this operation was performed. In addition to being an excellent procedure for low rectovaginal fistulas, the anal sphincter-preserving operation gives equally good results for correction of rectoperineal fistulas in males and females. Because this operation has received insufficient attention in the current medical literature, it seems desirable to report our experience with five patients in whom it has produced excellent functional results.

TABLE I
DISTRIBUTION OF IMPERFORATE ANUS ACCORDING TO TYPES

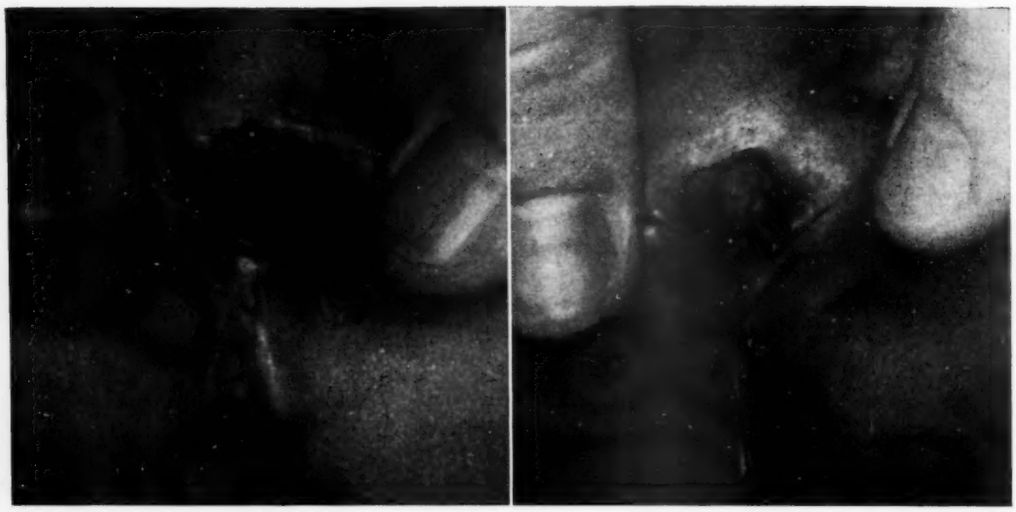
Type	Male	Female	Total
I	9	3	12
II	2	0	2
III	36	35	71
IV	2	1	3
Unidentified	0	1	1
	49	40	89

TABLE II
DISTRIBUTION OF PATIENTS BY SEX AND TYPES OF FISTULAS

	Male	Female	Total
Rectourethral	7	1	8
Rectoperineal	3	2	5
Rectovaginal	..	27	27
Rectovesical	4	2	6
Rectouterine	..	1	1
Rectoperineal and rectovesical	1	..	1
	15	33	48

INCIDENCE

About one in every 5,000 babies is born with an imperforate anus. Between 1937 and 1954 this diagnosis was made in sixty-nine of 954,928 admissions to Charity Hospital in New Orleans; and in twenty of 146,209 admissions to the Ochsner Clinic in the twelve-year period ending January, 1954. The distribution of these eighty-nine cases according to type is shown in Table I. Type III, the most common type encountered, comprised 79 per cent of the eighty-nine cases in our series. Thirty-two patients, or 36 per cent, had associated rectovaginal or rectoperineal fistulas (Table II), and it is in this group of patients that the anal



1A 1B

FIG. 1. A, photograph demonstrating location of most rectovaginal fistulas in the fossa navicularis; B, probe in fistula illustrating patency.

sphincter-preserving procedure is especially applicable.

LOCATION OF FISTULA

Rectovaginal fistula may open anywhere in the midline from the posterior fornix to the fourchet. Fortunately, most fistulous openings are located in the lower third of the vagina. In our twenty-seven cases only one opened into the upper third of the vagina, two in the lower third above the hymen, and the remainder in the fossa navicularis just below the hymen.

In the male the rectoperineal fistula may be found in the midline from the base of the penis to the anal dimple. In the female it may appear in the midline between the posterior fourchet and the anal dimple.

DIAGNOSIS

In the majority of cases rectovaginal and rectoperineal fistulas can easily be diagnosed by inspection. In some patients the fistulous opening is adequate for normal evacuation and with regular bowel habits may go undetected for many years. One of our patients with a rectovaginal fistula was twenty-three years old and married before the anomaly was discovered. In another, a six year old boy, a rectoperineal fistula was discovered during routine physical examination. Although many of these patients have regular bowel habits and get along fairly well, none has a controllable anus.

In some cases the diagnosis is not obvious. A small fistula may be difficult to find and in many instances the small opening may become plugged with the sticky, tenacious meconium causing complete intestinal obstruction. The possibility of an associated rectovaginal or rectoperineal fistula should always be kept in mind in patients with type III imperforate anus. Careful inspection of the fossa navicularis frequently will reveal a small dimple in the midline just below the hymen. (Fig. 1A.) Probing this area with a small ball tip silver probe or curved Kelly forceps will verify the existence of a fistula. (Fig. 1B.) Roentgenography following instillation of a radiopaque medium is usually unnecessary. If no fistula is detectable immediately after birth, oral feedings should be restricted and the patient observed for thirty-six hours. During this period obstruction will develop in infants with occluded undiagnosed fistulas and the gaseous pressure and distention of the rectum will frequently be sufficient to dislodge the meconium plug with evacuation of meconium and gas through the vagina or perineum. No harm will result during this period of observation, even if a fistula is not present. It should be emphasized that, after a period of twenty-four hours has elapsed, interpretation of roentgenograms made with the patient in the inverted and lateral positions with an opaque marker on the anal skin, as advocated by Wangensteen and Rice,[6] will be more accurate

FIG. 2. Infant immobilized by placing sacrum on an adhesive sling with buttocks protruding over edge. Lower extremities held in place with adhesive stirrups.

in determining the distance that separates the anal skin from the blind pouch of the rectum, so that the surgeon is better able to select the most appropriate surgical approach for correcting the existing anomaly.

PREOPERATIVE PREPARATION

Preoperative medication in preparation of the colon is similar to that currently employed in preparing patients for operations on the colon. A small polyethylene tube should be placed in one of the antecubital veins for replacement of blood loss and intravenous fluids during and after operation. The stomach should be decompressed by means of a small catheter prior to induction of anesthesia.

TECHNIC

Open drip ether or preferably a closed method,[7] if the apparatus modified for pediatric surgery is available, is used. Maintaining the position of such small patients may present a problem which we have solved by placing the patient on a taut strip of adhesive 4 inches wide. This acts as a sling several inches above the level of the table. From the center of the adhesive sling a T extension is directed toward the head of the table. The baby is placed on the adhesive in such a manner that the buttocks protrude over the edge, and the back and shoulders are secured to the upright portion of the T. The head is below the level of the buttocks as a prophylaxis against aspiration. The lower extremities are suspended upright with adhesive stirrups. (Fig. 2.) The surgeon is grateful for this complete immobilization of the patient during the long meticulous dissection.

Traction is made on each side of the vagina in order to keep the skin taut while a circumferential incision is made through the full thickness of the skin about the fistula. (Fig. 3.) The skin or vaginal mucosal edges of the fistula are grasped with several hemostats for traction, which is so necessary to accomplish the dissection. Sharp dissection is carefully carried out with scalpel and small scissors. After the fistulous opening has been mobilized, the rectum is separated from the vagina. The rectovaginal septum is extremely thin and the posterior vaginal wall at times seems to be no thicker than a sheet of paper. The dissection is kept close to the vagina, for although precaution is always being exercised to prevent buttonholing the vagina and rectum, making a hole in the vagina is associated with less serious complications than one in the rectum. During the proximal extension of this dissection the rectum is freed, the levator ani muscles being carefully separated from the rectum and retracted lateralward. After the levator muscles have been freed from the lateral aspects, the posterior aspect of the rectum is separated from the perineal body by sharp dissection. The greater part of the posterior rectal mobilization is accomplished with blunt finger dissection in the hollow of the sacrum. This dissection is extremely tedious, as it must be

meticulously anatomic. At times insertion of the index finger through the fistula into the rectum will greatly facilitate the dissection.

The rectum is mobilized to the cul-de-sac anteriorly and to a level just below the promontory posteriorly. (Fig. 4A.) After the prominent. A short anteroposterior incision about 1.5 to 2 cm. long is made in the midline over the anatomic center of the external sphincter (Fig. 4B), extending through the skin so as to preserve the external sphincter fibers immediately under the skin. We have

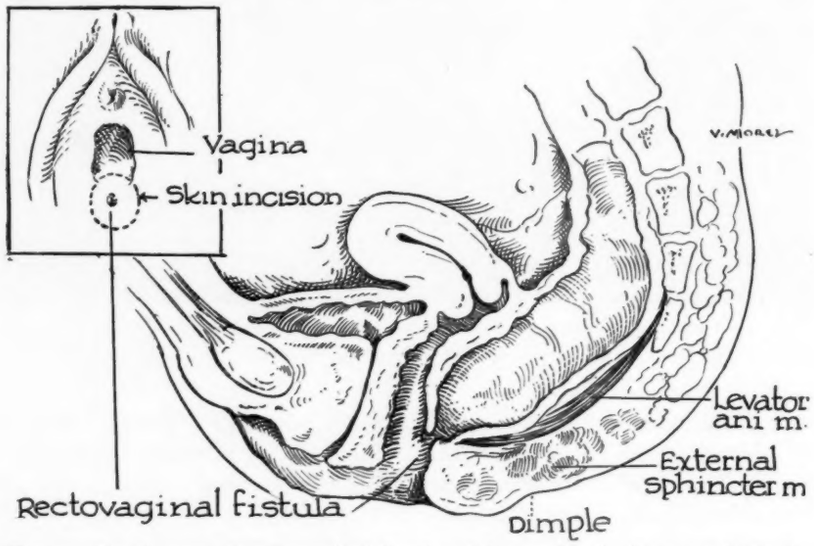


FIG. 3. Usual type of rectovaginal fistula with opening in fossa navicularis. Insert demonstrates circular incision employed in anal sphincter-preserving technic.

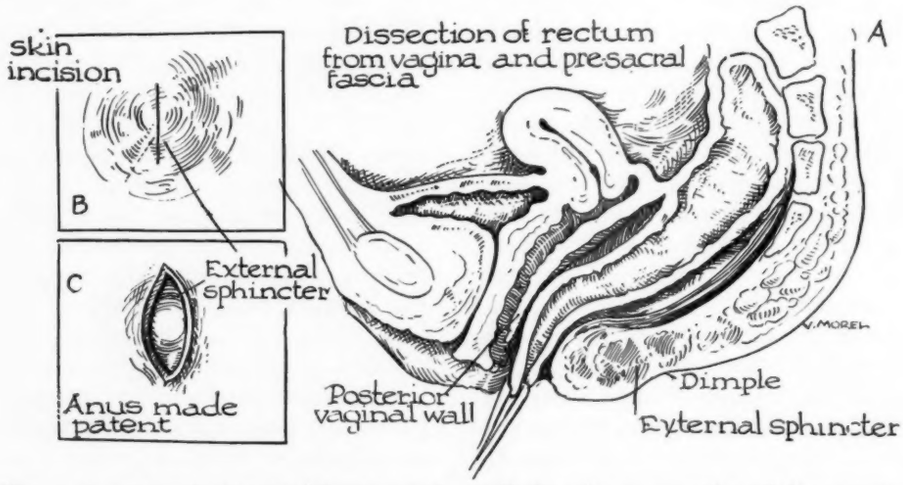


FIG. 4. A, rectum is separated from posterior wall of vagina to near the cul-de-sac anteriorly and posteriorly from the levator ani muscles and sacrum; B, anteroposterior incision in anatomic center of external sphincter through skin only; C, center of sphincter is made patent by spreading fibers with a hemostat and the mobilized rectum is pulled through an intact external sphincter.

rectum has been mobilized, the index finger is inserted through the operative wound superior to the perineal body in such a manner that pressure can be applied to the superior aspect of the levator ani muscles and anal sphincter. This maneuver makes the sphincter more found the external sphincter in most patients to be discoid in shape and non-patent, necessitating creation of a patent external sphincter by means of gradually stretching the centrally located muscle fibers outwardly by opening the blunt tip hemostat. (Fig. 4C.) The hemo-

stat is slowly worked through the center of the sphincter, which is dilated by repeatedly spreading the hemostat until the little finger can be introduced.

A curved Kelly forceps is inserted through the sphincter over the intact perineal body to grasp the mobilized rectum and pull it through the intact sphincter muscle. (Fig. 5.) The fistulous opening and the traumatized portion of the terminal rectum are amputated and the rectum is sutured to the anal skin with interrupted atraumatic chromic No. 000 sutures. (Fig. 5.) The sutures are placed only through the full thickness of the skin, care being taken to avoid inclusion of the underlying external sphincter muscle in anastomosing the rectum to the anal skin. The levator ani muscles are then coapted with interrupted cotton sutures between the rectum and vagina. (Fig. 5.) This separation of the rectum from the posterior vaginal wall is most important, for this not only constructs a thicker perineal body but also is a great factor in preventing retraction of the rectum. In patients with rectoperineal fistulas the transverse perinei muscles, in addition to the levator ani muscles, have to be sutured together. The defect in the vagina is loosely closed.

Drainage of the wound is not usually necessary. A small strip of vaseline gauze, placed into the rectum, will keep the line of anastomosis separated and thus prevent early stenosis by coagulation of the raw edges. There is no danger of retraction of the rectum or recurrence of the rectovaginal fistula if a good perineal body has been obtained, the rectum adequately mobilized and not injured during the procedure, and anastomosis performed without tension.

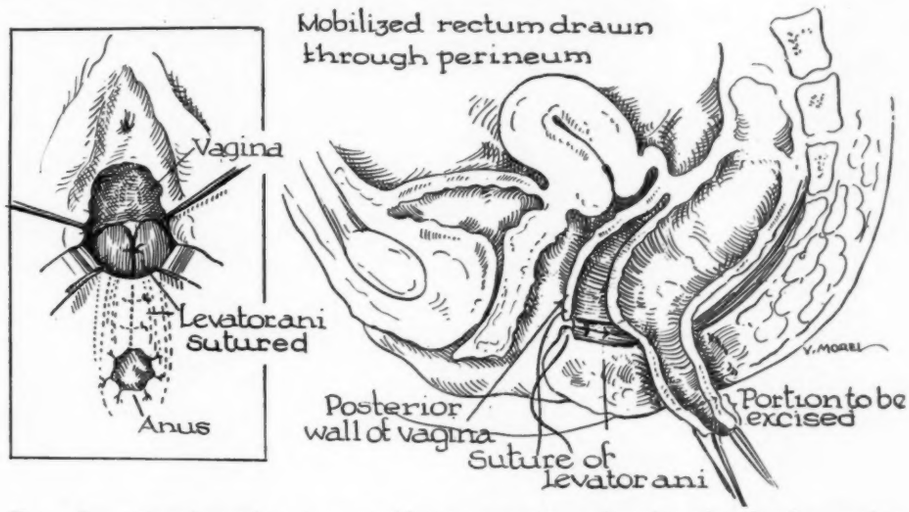
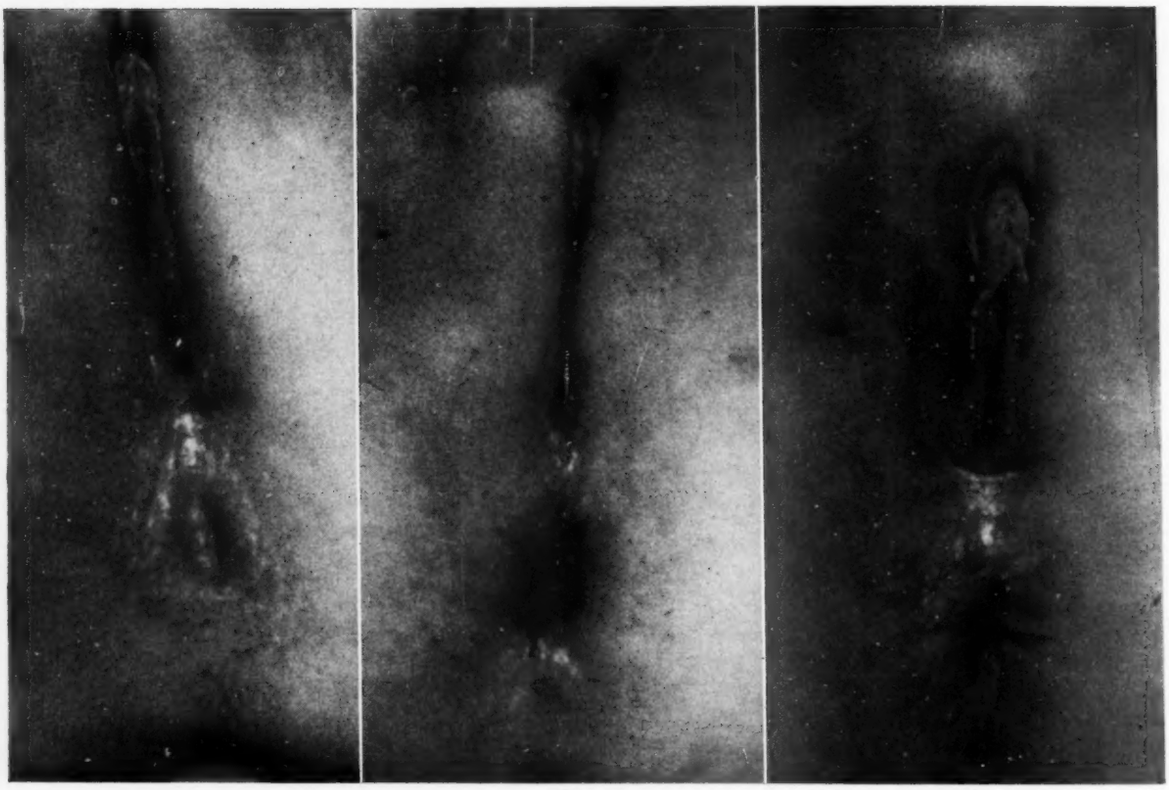


FIG. 5. Levator ani muscles are coapted between rectum and vagina; then fistular opening is closed. Insert illustrates suturing levator ani muscles through surgical wound at site of former fistula. After traumatized rectum is excised, the rectum is sutured to anal skin.

POSTOPERATIVE CARE

After nausea has subsided, the patient is given water, and on the following day his regular formula or diet. Sufficient mineral oil is given to prevent constipation during the first two weeks. The patient usually has a bowel movement on the second or third day. Administration of sulfathaladine and vitamin K is continued for about three weeks and penicillin and streptomycin for five days. The anus is kept clean by washing with cotton saturated with normal saline solution. Examination or dilatation of the anus is not done for three weeks, for every precaution must be taken not to disrupt the mucocutaneous anastomosis. After three weeks the wound has healed sufficiently to permit safe gradual dilatation of the anus with rubber catheters, followed by soft gum rubber dilators twice a day until the little finger can be inserted. The first few dilatations are done by the surgeon or resident surgeon. Only after the wound has healed and the anus admits the dilators is the patient discharged. The mother is instructed to use her little finger when the anus is large enough, and to continue to dilate the anus twice a day until the inflammatory tissue



6A 6B 6C

FIG. 6. Rectovaginal fistula. A, preoperative photograph showing anal dimple; B, photograph two days postoperatively; C, photograph eleven months postoperatively.

reaction is absorbed and the anastomotic scar is soft and pliable, which usually requires two to four months.

RESULTS

One of us (P. H. H.) has performed this sphincter-saving procedure on two patients with rectovaginal fistulas and three with rectoperineal fistulas. (Table III.) A para-anal abscess developed in one male patient with a rectoperineal fistula, necessitating incision and drainage, and later fistulotomy. This complication resulted from partial disruption of the line of anastomosis caused by rectal digital examination on the sixth postoperative day. In the female patient with a rectoperineal fistula the perineorrhaphy wound became infected, broke down and healed by secondary intention. This complication was attributed partially to use of chromic sutures. As a result, we now use fine cotton sutures. In spite of these two complications the surgical scar distortion of the anatomy was minimal, and the perineum and anus had a more normal appearance than in patients treated by the Rizzoli operation. Excellent results with a controllable anus were obtained in four patients. (Fig. 6.) The other patient has been operated on too recently to evaluate results but his progress thus far has been entirely satisfactory.

TABLE III
SUMMARY OF FIVE CASES OF IMPERFORATE ANUS TREATED BY ANAL SPHINCTER-PRESERVING OPERATION

Cases	Sex	Type of Fistula	Age at Operation (mo.)	Results
L. L.	F	Rectovaginal	4	Excellent
S. L.	F	Rectovaginal	8	Excellent
E. D.	F	Rectovaginal	51	Excellent
D. C.	M	Rectoperineal	5	Excellent
B. K.	M	Rectoperineal	64	*

* Too recent to evaluate.

COMMENTS

In no other surgical procedure is the final outcome more dependent on the success of the

initial operation. Because of the rarity of these anomalies, many of us lack the desired experience in their surgical correction.

Cases of imperforate anus, type III, with associated rectovaginal or rectoperineal fistula without obstruction are not considered surgical emergencies. Selection of the time for operation is dependent on a number of factors. In infants with sufficiently large fistulas to permit adequate bowel evacuation, it is best to wait until the patient is older, for the larger the patient the more successful the anatomic dissection will be. However, correction should be undertaken before the age of two years. We believe that plastic correction is indicated in all patients with this anomaly, in spite of apparent continence displayed by regular bowel habits and lack of difficulty with bowel movements.

In patients with extremely small fistulas the opening may be dilated with a rubber catheter or, if necessary, it may be snipped along the vaginal wall with pointed scissors for 0.5 cm. to relieve obstruction. If recurrent impaction results from inadequate bowel evacuation due to stenosis and enemas are ineffective, earlier operation is indicated. However, we do not believe infants with small fistulas should be operated upon immediately after birth because of some difficulty in bowel elimination. We prefer to wait two or three weeks, if possible, for this will permit the rectum not only to become dilated but also to descend further into the pelvis, thereby facilitating the surgical dissection. (Fig. 7B.) Moreover, this provides time to evaluate associated anomalies. Therefore, operation should be performed early only on patients who continue to have obstructive symptoms.

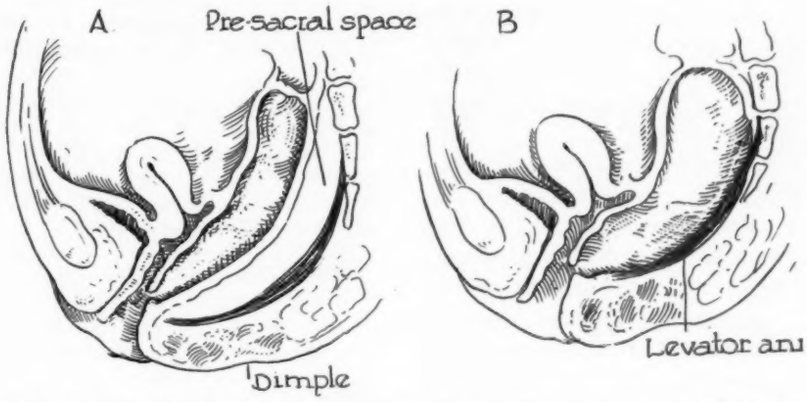


FIG. 7. A, in patients with a left colostomy because of rectovaginal fistula the rectum retracts and acquires a conical shape due to disuse. B, in patients without colostomy the rectum enlarges and descends further into pelvis.

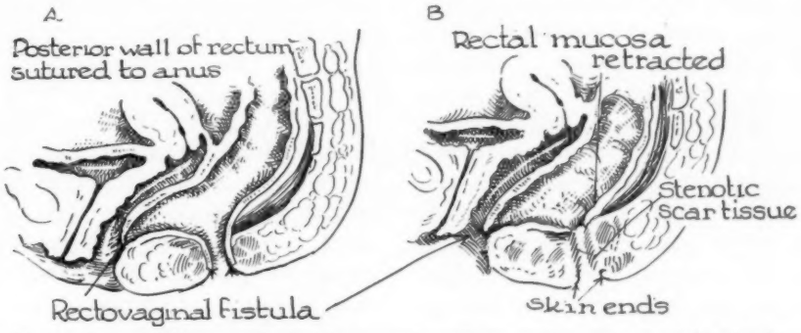


FIG. 8. A, not infrequently in obstructed undiagnosed rectovaginal fistula the posterior rectal wall is sutured to anal skin; B, stenosis because of retraction is a common complication in these instances.

It is desirable to delay Amussat's[8] perineal repair for imperforate anus, type III, for the first twenty-four or thirty-six hours of life, unless it is reasonably certain that an obstructed rectovaginal fistula does not exist. Immediate perineal Amussat repair in the

newborn with undiagnosed rectovaginal fistula usually results in incomplete mobilization of the rectum with anastomosis of the posterior wall of the rectum to the anal skin. This type of repair is associated with a high incidence of retraction of the posterior rectal wall because of fixation of the terminal end with resulting troublesome anal stenosis, which is difficult to correct. (Fig. 8A.) Moreover, it is highly improbable that a controllable anus can be obtained by surgical correction of this complication. (Fig. 8B.)

In these patients colostomy prior to anorectoplasty is not indicated. In patients seen for the first time with a colostomy continuity of the colon should be restored two to three weeks after the anorectoplasty, for dilation of the anus by bowel movements is the greatest prophylaxis against postoperative stenosis.

Absence of the external sphincter has been reported. Harken[9] believed that most, if not all, of these patients have an external anal sphincter. We have never seen a patient with an imperforate anus without an external sphincter. We have noted, however, that in patients without a well developed anal dimple or proctodeum the anal sphincter is a thin, flat, discoid non-patent muscle. In patients with a well developed proctodeum or anal dimple the external sphincter is well developed and the sphincter bundle is readily detectable and approaches more of the normal characteristics.

In our series all rectovaginal and rectoperineal fistulas did not have a true fistulous tract connection of the congenitally abnormally located anus to the rectum, but the fistulous opening joined immediately with a normal rectum. The lower rectum was not stenosed in a single case.

SUMMARY

A slight modification of a technic, originally described by Stone, is advocated for correction of low rectovaginal or rectoperineal congenital anomalies in which the external sphincter is preserved. Excellent functional results were obtained in four patients operated upon with this method; and in the fifth patient, operated upon too recently to evaluate the final result, an entirely satisfactory immediate result has been obtained. The advantages of this procedure over that of Rizzoli are that it does not necessitate severance of the perineal body and external sphincter, and the functional and cosmetic appearances approach those of the normal anus.

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DISCUSSIONS

John G. Matt (Tulsa, Okla.): It has only been eighteen years since Harvey Stone reported his modification of the Rizzoli procedure. I believe that this is more frequently used than the literature indicates. I have participated in at least eight such cases in my area, and I know of others that were similarly handled. I think that the lack of reports on the Stone modification is due to the relative novelty of this operation plus the fact that atresia ani vaginalis occurs so infrequently.

Parenthetically, I propose that some of our more senior members, who have used this operation ten or more years ago, conduct follow-up surveys and report to us their findings and conclusions.

The Stone technic uniformly yields excellent results as far as patency and continence are concerned, but it is frequently followed a few years later by mucosal eversion. A wet perineum develops in these patients because the artificial anal canal is composed of mucosa instead of the normal modified skin.

Dr. Vernon David, discussing Stone's classic paper, described how he formed skin flaps which are turned in from each side of the new anus and sutured to the mucosa forming a

skin-lined anal canal. This prevents anal ectropion. He describes his technic more completely in an article published in the February 1947 issue of *Surgery*.

I have succeeded in forming dry anuses by using a trifoil incision, centered over the anal dimple, undermining the triangles of skin so formed and suturing them to the cuff of vaginal mucosa so that as retraction occurs the flaps are pulled up to form a more nearly normal anoderm. The only merit my modification has is that it yields an undistorted anus as well as a dry one.

Dr. Hanley is to be congratulated for his lucid presentation of the Stone operation.

RAYMOND L. MURDOCH (Oklahoma City, Okla.): Numbers of observers have noted the occurrence of strictures and infrequently a turnout of wet mucous membrane following the type of operation described in this article. The same regrettable sequelae have occurred after some Whitehead-type hemorrhoidectomies.

In an attempt to remedy this I remembered the admonition to leave intact a longitudinal band of integument from skin continuous to the mucous membrane inside the rectum, and first did this in the congenital condition of atresia ani vaginalis in 1933. My article and case reports appeared with diagrams in the 1941 *Transactions of the American Proctologic Society*. The narrower longitudinal band used soon thereafter is better shown in the illustrations in the textbooks by Gabriel and Bacon (two-volume edition). You should more than halve the width of the band pictured for the Murdoch operation illustrated in the "Atlas of Operations of the Rectum and Colon" (by Bacon and Ross, 1954).

There is a great potential for dilation and stricture prevention in any longitudinal flap and band of integument that is continuous. If the rectum mobilized on three sides with its vaginal opening, falls naturally back to one side of the base of the U-flap, then the fairly narrow band of intact integument may spiral down in nearly a circle rather than be "accordioned" in a straight push back. After that opening is placed back in an approximately normal position, perineorrhaphy is all that is required to complete the operation.

Clinically many of these cases are not imperforate anus and are not atresia, but are a nearly normal, nearly continent anus in an abnormal location. The mucous membrane and bare bowel wall should not be skinned out of such, but the entire "doughnut" of the end (attached by the posterior flap) should be transplanted. I believe Dr. Matt and Dr. Hanley would agree that this was true in the patients upon whom they saw me operate.

The subcutaneous muscle that causes skin twitchings is in a sheath rather than a sphincter circle, when no lumen is present. This is true whether it be platysma of the neck, dartos of the scrotum; or in the intergluteal region. All of this sheath is preserved either in my narrow longitudinal band or at the sides of it. I do not see any advantage of dissecting in a tunnel to avoid incising an essentially cutaneous perineum, when hundreds of successful perineorrhaphies are being performed.

The intact posterior longitudinal flap with the narrow U-incision is one additional factor against stricturing. I indicated in my original paper that this might be more important in the younger than in the older patients. I think they all should have complete transposition operation by two or three years of age.

PATRICK H. HANLEY (closing): I am very grateful to Dr. Matt and Dr. Murdock for their discussion. I wish to thank Dr. Murdock for extending to me an invitation to watch him do his operation. I must say that he makes the procedure sound technically easy to perform. I am sure that his procedure has proved effective in many hands.

Leiomyosarcoma of the Rectovaginal Septum

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Smooth muscle tumors of the gastrointestinal tract are uncommon, and among the least common of these is the malignant smooth muscle tumor of the rectum. In our search of the literature we have been able to find only eighteen cases. Because of its rarity it is deemed worth while to report a case of leiomyosarcoma of the rectovaginal septum in which there was repeated and severe rectal bleeding.

Sarcoma is the rarest rectal tumor, and as a group is characterized by slow growth, local recurrences, rare lymph node metastasis and death from vascular involvement with metastasis. The most significant pathologic characteristic of sarcoma is its local invasiveness. Blood vessels are always invaded and eroded, and as a result tumor emboli and hemorrhage are likely to occur.

In a typical case of sarcoma of the rectum the tumor is relatively asymptomatic and produces its earliest symptoms because of its space-consuming and polypoid character. The tumor begins as a small submucosal nodule and extends into the rectum. It is difficult to detect the growth in its early stage by proctoscopic examination, as no bulging or ulceration has formed.[1]

CASE REPORT

C. S., a forty-one year old Negress, entered the Los Angeles County Hospital in May, 1953, complaining of pain around the anus of six hours' duration. She had no other complaints at the time but on subsequent questioning admitted to occasional mild dyspareunia during the preceding six months. Some of the more significant subjective symptoms described by the patient were a feeling of fullness and heaviness associated with a bearing-down sensation in the perineum. In addition she complained of incomplete evacuation after each defecation, with a difficult passage of ribbon-like stools. In April, 1944, at another hospital she had had a subtotal hysterectomy for a multilobular uterine tumor which on gross and microscopic examination proved to be fibromyoma.

On rectal examination a firm, moderately tender, extrarectal globular mass about 3 inches in diameter was palpated beneath the anterior wall of the rectum beginning immediately above the mucocutaneous junction. The tumor occluded approximately one-half of the lumen of both the rectum and vagina but was not attached to the mucosa of either. The lesion was noted to be sharply separated from the surrounding tissues of the rectovaginal septum.

The blood pressure was 120/70 mm. mercury. The remainder of the physical examination was negative.

Laboratory data were as follows: hemoglobin, 11.0 gm. per 100 cc.; white blood count, 12,800; urinalysis, negative; non-protein nitrogen, 22 mg. per 100 cc.; CO_2, 30 mEq. per L.; chlorides, 101 mEq. per L.; sodium, 135 mEq. per L.; potassium, 7 mEq. per L.; icteric index, 5; albumin, 4 gm. per 100 cc.; globulin, 2.4 gm. per 100 cc.; cephalin flocculation test, negative in 24 and 48 hours; thymol turbidity, 1; prothrombin time, 135 per cent on control; serology, negative; Frei test, negative.

The electrocardiogram was within normal limits and roentgen ray of chest and abdomen revealed no pathologic findings. Punch biopsy of the rectal tumor showed leiomyosarcoma of the rectum, low grade.

Following punch biopsy brisk bleeding was noted within the substance of the tumor, associated with an abrupt increase in its size with virtual occlusion of the rectal lumen. The mass was intentionally incised through the rectum to extract a large amount of the fluid and clotted blood. The resulting cavity was firmly packed with oxycel® gauze to control the profuse bleeding that developed after partial evacuation of its contents. Despite the cautious packing the patient continued to bleed freely; this increased gradually to hemorrhagic proportions and required repeated transfusions to a total of 2,500 cc. during the three-day

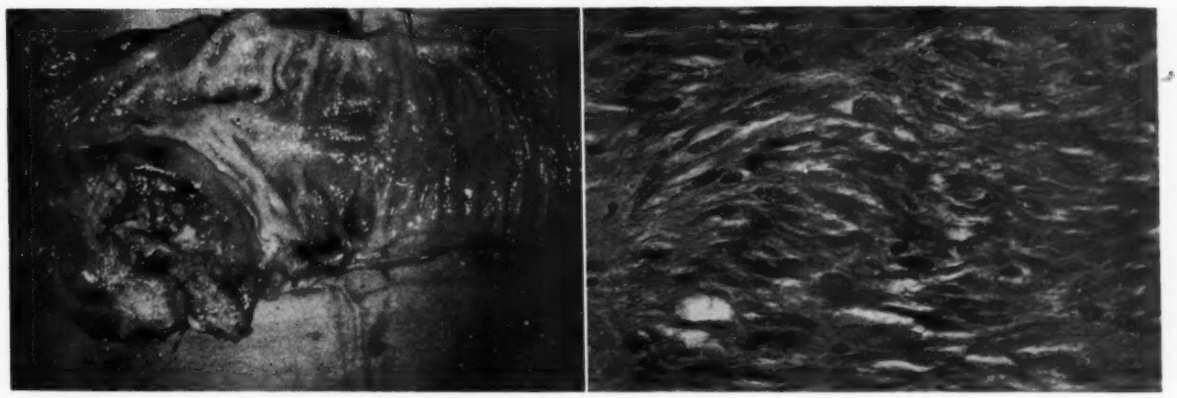

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FIG. 1. Approximately 1 cm. above the mucocutaneous junction a fairly well circumscribed ovoid mass bulged into the rectal ampulla. It was covered with normal rectal mucosa except for a small defect. A thin capsule separated the mass from the vaginal septum.

FIG. 2. Showing interlacing bundles of spindle-shaped cells interspersed with mitotic figures which are consistent with leiomyosarcoma.

period. Additional packing was also inserted into the cavity of the tumor mass to control the bleeding. After adequate preparation of the bowel with succinyl sulfathiazole and terramycin,® an abdominoperineal resection was performed. No enlarged glands were palpated and no liver metastases were found. The tumor was readily separated from the vaginal wall. The postoperative course was uncomplicated and the patient left the hospital on the fourteenth postoperative day in good general condition.

Pathologic findings were as follows: At macroscopic examination of the specimen removed at operation in June, 1953, a segment of bowel was seen which was 22 cm. long, previously fixed in formalin. The distal surgical margin was anal skin. About 14 cm. from the proximal surgical margin and 1 cm. from the anal skin there was a mass 5.5 cm. long by 7 cm. wide and 3 cm. thick. On the mucosal surface of the mass there was an ulcerated area 3 cm. long by 2.5 cm. wide and 2 cm. deep which had rolled mucosal edges for one-half its margin and tumor tissue for the other. The tumor extended to the adventitial surface but grossly appeared encapsulated by a thin membrane. The microscopic diagnosis was leiomyosarcoma of the rectum. (Figs. 1 and 2.)

COMMENTS

Leiomyoma of the rectum may arise from three strictures: (1) the smooth muscle of the rectal wall, the muscularis mucosa, (2) the circular and longitudinal muscle layers and (3) the walls of the blood vessels. The tumor is composed of smooth muscle occurring very infrequently and developing into various sizes, some of which become exceedingly large, either in single or multiple form. Its consistency is usually firm or hard with a smooth surface.

The first report describing myoma of the rectum was made by Vander Espt in 1881 (Hunt[2]). Myomatous lesions occurring in the upper gastrointestinal tract are in the majority of cases of a benign nature, and only 50 per cent of those located in the colon and rectum were found to be malignant.[3] Newman[4] reported that malignant leiomyoma of the rectum had been noted in only eight prior cases. The existence of low grade malignancy in leiomyosarcoma was noted by various authors.[3,5,6] From a study and review of the cases reported it appears that regressive changes in myomatous tumors cause a softening or disintegration of a part of the neoplasm. However, the rate of growth may become so rapid the tissue assumes an embryonic type undistinguishable from smooth muscle.

Even though metastasis is late in these slowly growing tumors, local excision of leiomyosarcoma is almost invariably followed by recurrence, regardless of grade. Roentgen ray treatment is of very doubtful value. The only adequate approach for treatment of leiomyosarcoma of the rectum is radical resection.

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Ureteroileal Sigmoidostomy

CONTROL OF URETERAL REFLUX IN URETEROSIGMOID ANASTOMOSIS

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WHEN the lower urinary tract is excised or is no longer capable of maintaining urinary flow, the management of one or both ureters becomes a challenging problem. This situation is met with increasing frequency as the limits of radical surgery are extended and pelvic evisceration becomes more frequent. Even when such lesions as carcinoma of the cervix and bladder are incurable, strategically placed local extensions may produce low ureteral obstruction, uremia and death while the disease is still confined to the pelvis. Proper reconstruction to maintain urinary flow may give gratifying palliation.

If the sigmoid and rectum are not available for transplantation of the ureters, cutaneous ureterostomies, a wet colostomy or a substitute bladder may be utilized. An isolated ileal segment to which both ureters have been transplanted serves adequately as an artificial bladder. The distal end of the ileal segment is brought through the abdominal wall as in an ileostomy. Urine is collected in an ileostomy bag with satisfactory result. Cutaneous ureterostomies and wet colostomies are burdensome to the patient.

Ureterocolonic anastomoses are usually done when the sigmoid, rectum and anal sphincter mechanism are intact and ureters must be transplanted. Evaluation of this procedure is difficult. When carried out in children for exstrophy of the bladder and in adults for benign disease, many patients live long, comfortable lives. However, it is becoming increasingly clear that when this type of transplantation is done for malignant disease, diversion of urine into the bowel yields poor results in terms of patient comfort and longevity.[1]

The long-term satisfactory result of ureterocolonic anastomosis depends on the absence of ascending infection and maintenance of an unobstructed urinary flow. In the case of a ureterosigmoid anastomosis, obstruction to the urinary flow may result from purely physiologic factors. Baker[2] found that in animals intracolonic pressures exceeded those within the ureter even before the defecation reflex was initiated. This suggests that unless the colon is emptied quite frequently obstruction to the flow of urine and reflux of colonic contents up into the ureter is inevitable. This reflux results in recurring pyelonephritis, gradual destruction of the kidney, uremia and death.

Two methods are in general use for ureterosigmoid anastomosis. In one, emphasis is placed on the construction of a submucous tunnel (of variable length) in the bowel wall through which the ureter passes before entering the lumen of the intestine. Supposedly the conduction of feces toward the rectum exerts pressure against the ureteral opening, thereby producing a valve-like action.[3] Postoperative stenosis with poor drainage and subsequent hydronephrosis frequently follows this use of the tunnel technic. In the second method, direct mucosa-to-mucosa anastomosis is done. Theoretic conceptions in this regard suggest that a mucosa-to-mucosa anastomosis is less prone to stricture formation and subsequent obstruction. From a practical point this is most certainly true; this type of anastomosis not only remains patent, but also the ureter often becomes a diverticulum of the sigmoid full of gas and feces. In these instances the propulsive force of the ureter is not strong enough to present an impediment to the retrograde passage of colonic contents into the ureter.

During the study of isolated ileal segments serving as substitute bladders an attempt was made to visualize one of these pouches by x-ray after filling it with diodrast. Surprisingly, it was found that the intense peristaltic activity of the loop prevented the diodrast® from being retained long enough for a satisfactory film to be made.

In part, this intense peristaltic activity of

the loop may be explained by a hypothesis suggested by Bricker[4] that a critical level of hydrostatic pressure in these segments provides the stimulation for evacuation of the fluid.

With these observations in mind, an attempt

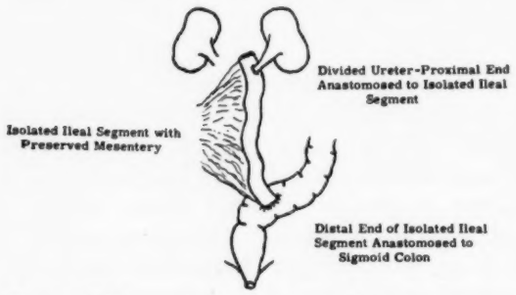

Divided Ureter-Proximal End Anastomosed to Isolated Ileal Segment
Isolated Ileal Segment with Preserved Mesentery
Distal End of Isolated Ileal Segment Anastomosed to Sigmoid Colon

FIG. 1. Diagrammatic representation of ureteroileal sigmoidostomy.

was made in the following case to utilize a new method of diverting the urinary stream into the colon in hopes that less reflux and ascending infection might result.

CASE REPORT

U. I., a fifty-eight year old white woman, was treated for carcinoma of the cervix with x-ray and radium during 1951. She was admitted to City Hospital on December 26, 1952, because of general weakness. Examination revealed marked fibrosis and immobility of the pelvic organs. The blood urea nitrogen was 170 mg. per 100 cc. and the CO_2 combining power was 52 volumes per cent. On the fifth hospital day a left nephrostomy was done and on the tenth hospital day a right nephrostomy was performed. By the twenty-fifth hospital day the blood urea nitrogen had dropped to 33.6 mg. per cent. The right nephrostomy tube failed to drain any urine and was removed on the fifteenth hospital day since it was thought that the right kidney was completely destroyed.

On the twentieth hospital day an exploratory laparotomy was performed. Metastatic carcinoma was present in the liver and there was widespread local extension of tumor, making pelvic evisceration unfeasible. Both ureters were greatly dilated with obstructing foci of tumor in the terminal portions. For palliative purposes, to avoid the discomfort of a permanent nephrostomy, a left ureteroileal-sigmoid anastomosis was performed. (Fig. 1.) This was accomplished by anastomosing the end of the left ureter to the proximal side of

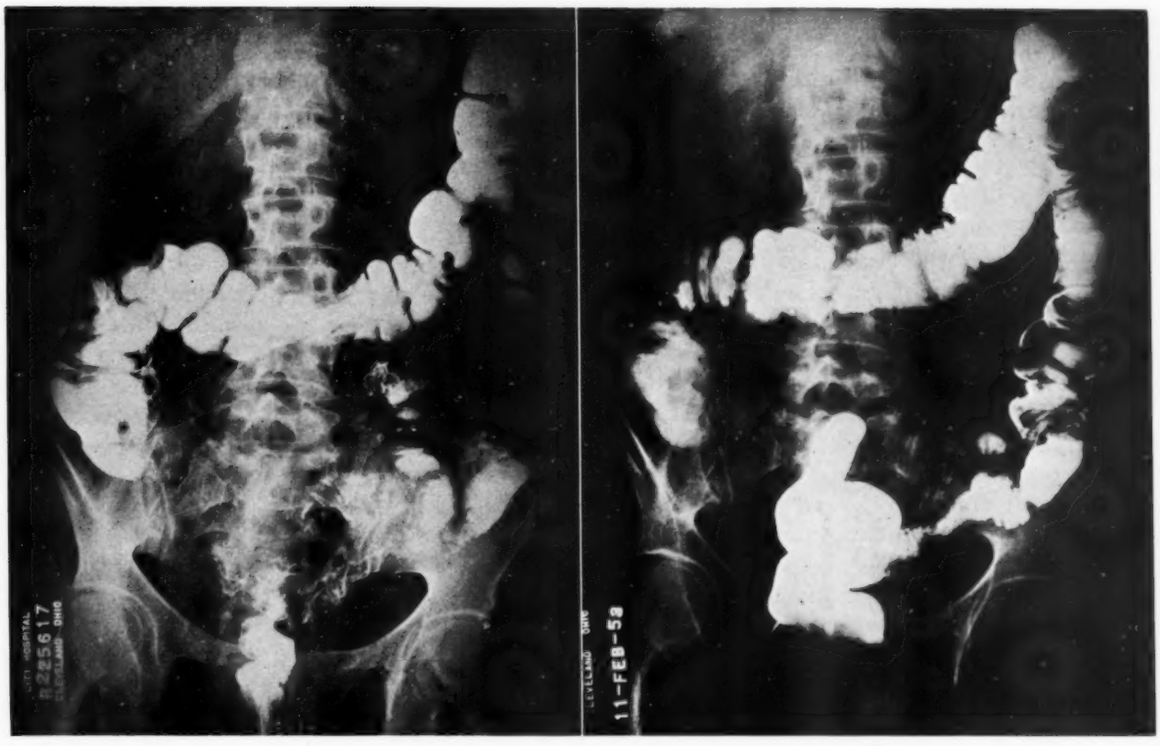
FIG. 2. Intravenous pyelogram made on the forty-fourth day showing fair function in the left kidney.

an isolated ileal segment and anastomosing the distal end of the isolated ileal segment to the side of the sigmoid. Two-layer anastomoses were done with a mucosa-to-mucosa layer of interrupted arterial silk first followed by a layer of fine catgut. On the third postoperative day the left nephrostomy tube was removed and the patient began to have six to eight watery bowel movements daily. Urine ceased to drain from the nephrostomy wound on the eleventh postoperative day and the patient was discharged on the forty-fourth hospital day. Intravenous pyelograms at that time revealed fair function in the left kidney. (Fig. 2.)

On March 18, 1953, the patient was readmitted in a moribund condition and died the following day. Postmortem examination revealed squamous cell carcinoma of the cervix with widespread local extension and remote metastases. Both kidneys showed evidence of pyelonephritis with abscess formation. The ureteroileal-sigmoid anastomosis was patent.

COMMENTS

An attempt to reduce ureteral reflux experimentally in dogs has been reported by Rieger and Harris.[5] These authors anastomosed the



3A 3B

FIG. 3. A and B, reproduction of x-ray taken during barium enema done under pressure showing evidence of slight reflux into distal ileal segment of ureteroileal sigmoidostomy.

dome of the bladder to an isolated segment of terminal ileum and then anastomosed the distal segment into the sigmoid. In the presence of an intact vesicoureteral mechanism evaluation of ureteral reflux is difficult. Mucosa-to-mucosa anastomosis between the ureter and small bowel in the dog was attempted by us in a series of ten dogs, but because of the small size of the dog ureter we were unable to develop a leak-proof anastomosis. No report is available of ureteroileal-sigmoid anastomosis in a human patient.

The existence of a gradient of peristalsis in the gastrointestinal tract has long been known. This gradient works effectively to transmit urine from the kidney to the bladder when a segment of ileum is substituted for the ureter. In this case attempts to force material up into the ureter during the course of a barium enema done with increased pressure showed filling only of the distal segment of isolated ileal segment. (Figs. 3 and 4.) This suggests that if the segment of ileum is properly interposed between sigmoid and ureter, the reconstruction may well function as a physiologic valve. In addition, since distention of the ileal segment stimulates its evacuation, partial reflux of the distal portion of the segment with feces may well stimulate its rapid evacuation from above downward.

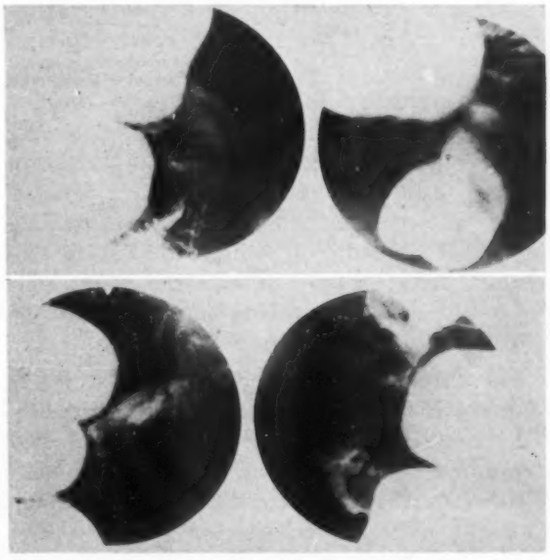


FIG. 4. Spot films made during course of barium enema showing anastomosis between ileal segment and sigmoid.

Ureteroileal-sigmoid anastomosis as described herein also allows for a mucosa-to-mucosa anastomosis at both the ureteroileal junction and ileosigmoid junction. The advantages of this type of union have been mentioned previously.

When the ureters are anastomosed to ileal segments, very little mobilization of the ureter is required because of the great mobility of the ileal segment and of its mesentery.[9] This problem of mobilization of the ureter is especially troublesome on the right side.

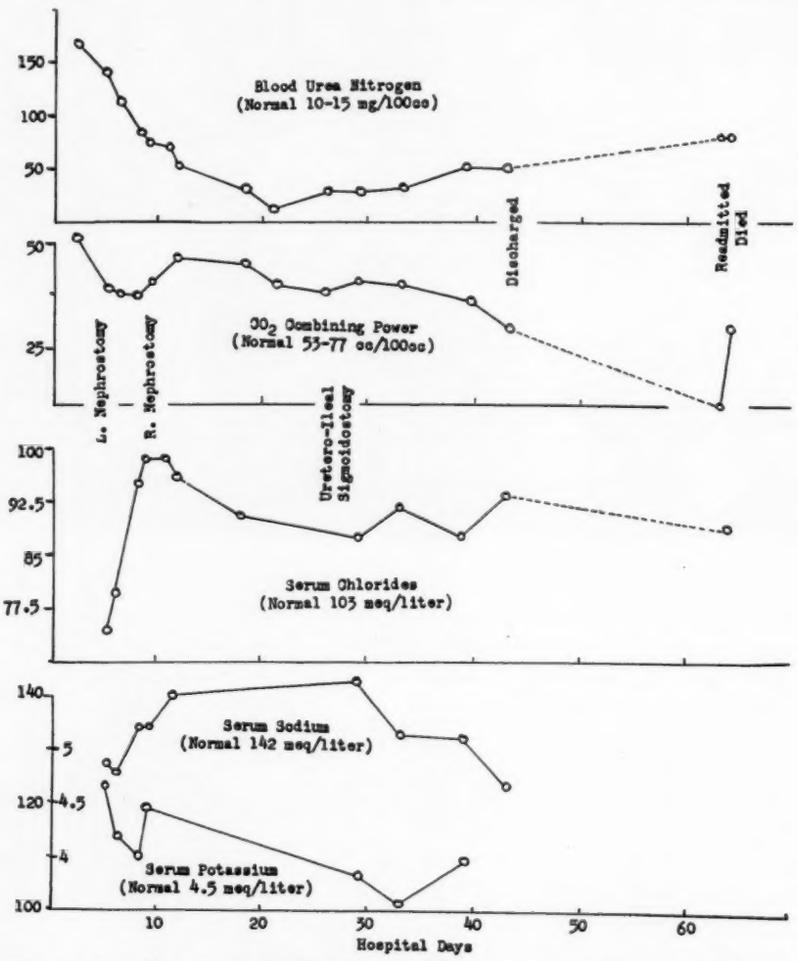


FIG. 5. Graphic representation of blood and serum chemical values during period of study.

The chronic pyelonephritis which seems invariably to result following ureterosigmoidostomy has been attributed in part to infection ascending from the colon via the lymphatics of the ureter.[8] Interposition of the ileal segment may well eliminate this factor.

Another technical problem not infrequently present in transplantation of ureters is the mobilization of the ureter. If the ureter is mobilized for any distance and displaced from its bed, its extrinsic blood supply, which is of a segmental nature, will be damaged. This may result in inflammatory edema and later fibrosis and stricture at the ureterosigmoid junction.

The problem of abnormal absorption of nitrogenous products in this type of ileal segment has not been noted. Long segments of ileum have been interposed between ureter and bladder in patients, and abnormal absorption has not been detected.

Although the survival period following operation in the case presented was quite short, this was to be expected in view of the advanced state of kidney disease present at the time of operation. (Fig. 5.) The operation itself was intentionally utilized in this type of patient since it was considered an experimental approach. The results, however, indicate that the

operation is feasible and its use in better risk patients may well be justified.

SUMMARY

Basic problems inherent in ureterosigmoid anastomosis are discussed. A case report is presented describing the use of ureteroileal sigmoidostomy in a patient with far advanced carcinoma of the cervix with ureteral obstruction as a palliative maneuver to avoid permanent nephrostomy. The possibility of utilizing this type of anastomosis to decrease colonic ureteral reflux and ascending infection when the urinary stream is diverted to the colon is discussed and its merits considered.

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DISCUSSION

Malcolm R. Hill (Los Angeles, Calif.): My remarks on this very interesting paper are along the complimentary line. I am happy to know that we have younger members in this organization who have a surgical background and who have the intestinal fortitude to go into this field. Every proctologist's office should be a cancer detection center. Dr. Rack has taken us beyond the realm of cancer detection; he has taken us into the realm of prolongation of life.

There is a Biblical phrase which says in effect that if an eye offend you, take it out. Certainly in our methods of approach to cancer in this era in which we are living we have to follow that Bible instruction to the "nth" degree.

Where you can draw the line on what you should and should not do is a $64 question.

I received a letter in which the patient's sentiments were expressed in these words: "Dear Doctor: I am working five days a week. Here is hoping that you live longer than I do so you will be around to take care of me!"

This patient had one of the largest rectal growths that I have ever removed. It was done nineteen years ago. One-tenth of the bladder wall was involved and transplantation had to be carried out on one ureter. It was the urologist who referred me to the patient and assisted me. We were criticized at that time for the time element involved; those were the days before antibiotics. This man had complete obstruction the day before we operated. I was desperate and tried to thread a catheter through his stricture. He let out a war whoop while in the inverted position and asked to be let up quickly. I allowed him to go to the toilet and he completely decompressed through his urinary bladder.

Now if that man can keep going nineteen years, here's hoping that Dr. Rack in his ureterosigmoid anastomosis procedures carries us into the realm of that patient who has a widespread involvement; and whether or not an elaborate procedure is employed, let us hope that more patients will be alive at nineteen years with radical procedures of this type.

I would like to ask Dr. Rack a question: Would you recommend ureteroileosigmoidostomy in the individual upon whom we have had to perform an abdominal-perineal proctosigmoidectomy with perineal colostomy?

Frank J. Rack (closing): We have not done wet colostomies as they are extremely troublesome to patients. We do our colostomy on the right side, with an ileal pouch on the left side to which both ureters have been transplanted. That type of patient is much more satisfied.

Explosions during Lower Bowel Electrosurgery

A METHOD OF PREVENTION

EDWARD I. LEVY, M.D., *San Diego, California*

MANY proctologists have encountered gas explosions in the colon during electrosurgery. Such an explosion occurred two years ago while I was performing electrodesiccation of a benign rectal polyp. Knowing that these explosions occur by ignition of inflammable colonic gas, I started experimenting with various gas mixtures to determine the most efficient method of prevention of colonic gas explosions. It is my purpose to outline these experiments and to present a simple and practical technic that has been proved satisfactory in my hands.

TABLE 1
COMPOSITION OF INTESTINAL FLATUS
(Percentages Found in a Review of the Literature)

Intestinal Gas Found (%)	CO_2	O_2	CH_4	H_2	N_2
Highest	25.4	20.0	44.0	43.9	87.0
Lowest	00.0	00.0	00.0	00.0	24.7
Average (Kirk[5])	9.0	3.9	7.2	20.9	59.0

Volume of flatus average 1.48 ml. per minute

The danger of gas explosions in the bowel during electrosurgery has been reported by Armous,[1] Becker,[2] Carter,[3] Lieberman[6] and Montier.[7] The resultant injuries may vary from ecchymosis to multiple ruptures of the bowel. Undoubtedly, these reports do not reflect the true incidence of these mishaps. Many of us who have encountered such explosions have not reported them.

COMPOSITION OF INTESTINAL GAS

Gas in the intestines, according to Wangensteen,[9] is produced by: (1) digestive processes, (2) bacterial fermentation, (3) diffusion of gas into the bowel from the blood stream and (4) swallowed air.

Ruge, Kirk,[5] Fries and others have analyzed intestinal flatus. Carbon dioxide, oxygen, nitrogen, hydrogen and methane were found in varying amounts. Ruge (cited by Lieberman[6]) found that on a milk diet hydrogen may be increased to 43.9 per cent. On meat or legume diet hydrogen is decreased and methane is increased up to 44 per cent. (Table 1.)

Since hydrogen and methane may be present in the bowel in high concentrations, an extremely explosive gas could be present during electrosurgery.

PROPERTIES OF EXPLOSIVE GAS MIXTURES

Combustible gas when mixed with certain proportions of air (or oxygen) will explode when ignited. The least concentration of gas; mixed with air, that will explode is called "the lower explosive limit." Concentration of combustible below this point will not explode. If the concentration of combustible in the mixture is progressively increased, a composition will be reached at which point the mixture is again non-explosive. Concentration of combustible just before this point is reached is called "the upper explosive limit." Inert gases such as carbon dioxide or nitrogen will narrow the explosive range. When inert gas is mixed in suitable proportions, explosion can be prevented. (Fig. 1.)

A study of Figure 1 shows the lower explosive limit of hydrogen in air to be a concentration of 4.10 per cent hydrogen. The upper limit is about 74.00 hydrogen in air. As an increasing amount of inert gas, such as carbon dioxide, is added to the mixture, the lower explosive limit is raised and the upper limit is depressed. The explosive range is thus narrowed as shown in Figure 2.

PURGING TO PREVENT GAS EXPLOSIONS

Gas chemists in industry use "purging principles" as a means of avoiding gas explosions in storage tanks and gas mains during repairs. This may be done in part by: (1) complete dis-

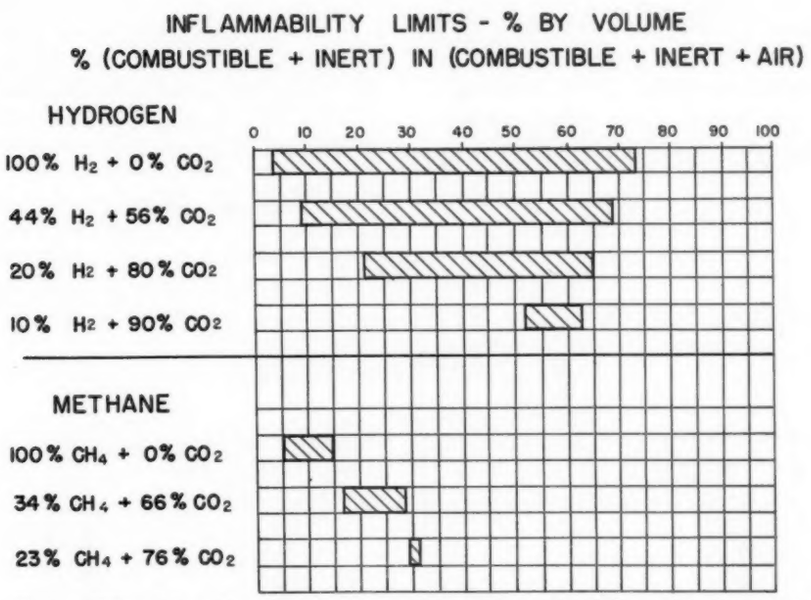
INFLAMMABILITY LIMITS - % BY VOLUME
% (COMBUSTIBLE + INERT) IN (COMBUSTIBLE + INERT + AIR)
HYDROGEN
100% H2 + 0% CO2
44% H2 + 56% CO2
20% H2 + 80% CO2
10% H2 + 90% CO2
METHANE
100% CH4 + 0% CO2
34% CH4 + 66% CO2
23% CH4 + 76% CO2
0 10 20 30 40 50 60 70 80 90 100

FIG. 1. Effect of carbon dioxide on repressing the explosibility of hydrogen or methane in air.

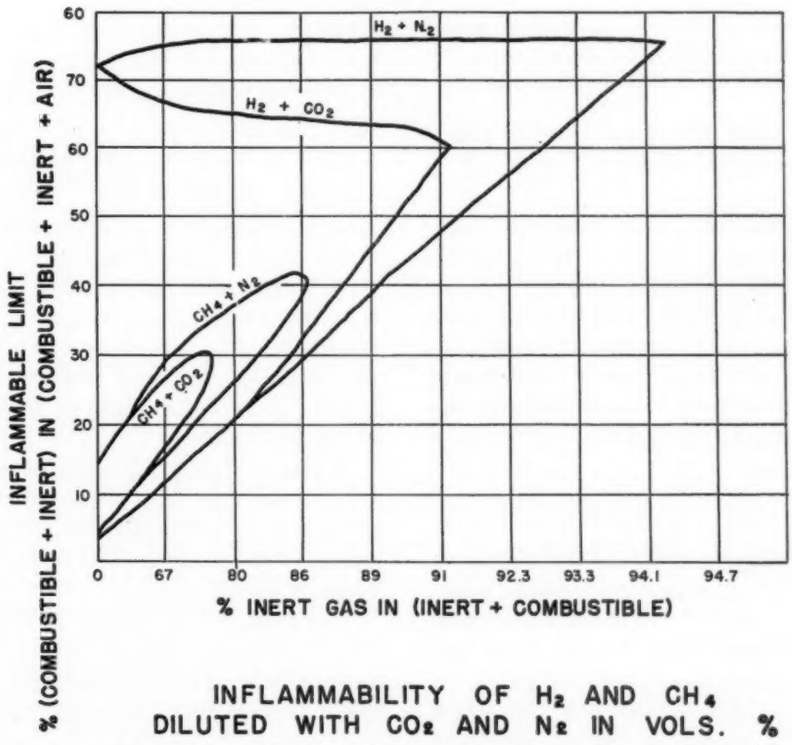

FIG. 2. Inflammable limits for hydrogen and methane diluted with carbon dioxide or nitrogen.

placement of combustible gas with carbon dioxide or nitrogen; (2) mixing inert gas with a combustible mixture in the proper amount to prevent an explosive mixture; (3) by placing a barrier of inert gas between the inflammatory gas and the ignition source. The last two methods appear to be the most feasible application of the "purging principle" applied to the colon.

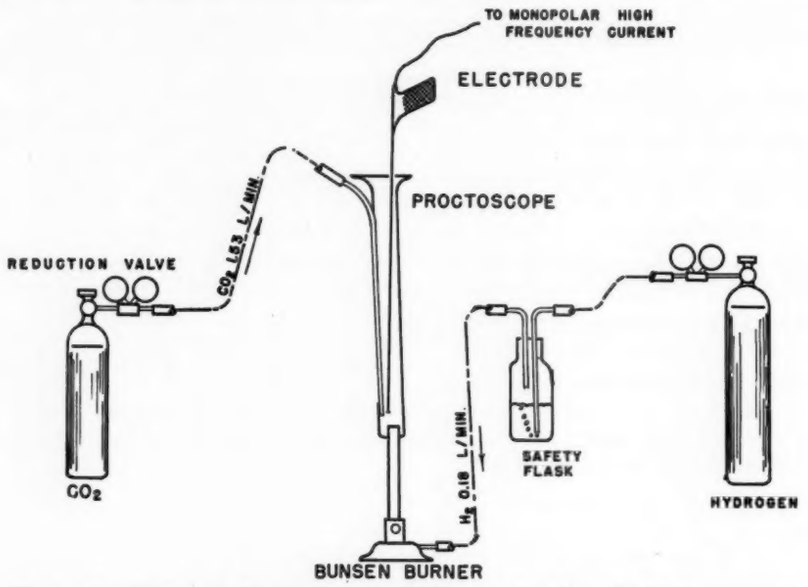

FIG. 3. Apparatus used in testing the efficiency of carbon dioxide in preventing explosions of hydrogen and air within a sigmoidoscope.

Procedures such as electrosurgery, performed in the presence of combustible gases, should be carried out in atmospheres which are outside the limits of inflammability of the gases involved. Preferably, the concentration of combustibles should be kept below the lower explosive limit. This may be achieved by the addition of sufficient quantities of carbon dioxide or nitrogen.

EXPERIMENTAL STUDIES

Hydrogen and air was used as the explosive test mixture. Hydrogen was used because it has a wider range of explosive limits than methane. Carbon dioxide was utilized as the inert gas since it is heavier than air. It will diffuse down the proctoscope and proximally into the bowel when the patient is placed in an inverted position.

The end of a sigmoidoscope was held over a Bunsen burner. The burner was connected to a source of hydrogen with a constant flow of .18 L. per minute. The sigmoidoscope was allowed to fill with hydrogen and air. An electrode, connected to a high frequency monopolar current and foot switch, was then inserted into the sigmoidoscope. The explosive mixture was ignited by passing a spark between the electrode and Bunsen burner or wall of the sigmoidoscope. On each ignition, there was a small explosion. This situation parallels the condition that may be found in the bowel just prior to a gas explosion during electrodesiccation.

An electrode equipped with a suction tip was then substituted for the plain electrode. Carbon dioxide, at the rate of 2 L. per minute, was blown into the electrode at the place designed for suction. Ignition was again attempted. Sparking caused explosions in over 75 per cent of the ignitions.

A Buie fulgurating sigmoidoscope, equipped with a suction tube, was then used. Carbon dioxide was introduced through the tube designed for suction. The plain electrode was again utilized. (Fig. 3.)

It was found that with a minimum constant flow of carbon dioxide of 1.53 L. per minute passing into the sigmoidoscope, the explosions could be prevented in 98 per cent of the trials.

EXPLANATIONS OF RESULTS

In these experiments, it required about nine volumes of carbon dioxide to prevent explosion of one volume of hydrogen in air. The failure of the electrode blowing carbon dioxide into the

explosive mixture was due to two main causes: (1) the outlet holes at the sparking tip were too small to permit an adequate flow of carbon dioxide; (2) the holes, being placed at the side of the electrode, divert the flow at right angles instead of toward the spark. Conversely, the efficiency of the sigmoidoscope in diluting the explosive mixture was due to the larger tube permitting a greater flow of carbon dioxide. The direction of the flow was toward the spark.

The ideal situation is to envelop the spark with a steady stream of carbon dioxide during electrosurgery.

COMMENTS

Armous[1] in 1945 described an anoscope especially constructed to permit the use of carbon dioxide during electrosurgery. He did not present any experimental data, but recommended 2 L. per minute flow of carbon dioxide. This corresponds with the flow of carbon dioxide I found to be adequate.

Other methods recommended to prevent colonic explosions are: colonic lavage, continuous suction and the use of tampons. These do not appear logical. Explosions have been reported following the use of lavage and tampons. An enema may increase peristalsis and cause a sudden rush of flatus into the distal bowel during electrosurgery.

Becker[2] in 1953 advocated the use of carbon dioxide in the prevention of colonic explosions. He also discusses other safety measures to be used in electrosurgery.

PRACTICAL APPLICATIONS

Any sigmoidoscope, equipped with a smoke suction tube, may be employed. A tank of carbon dioxide, fitted with a reduction valve and a liters-per-minute gauge, is used as a source of inert gas. Carbon dioxide is carried to the sigmoidoscope by means of rubber tubing of at least 5 to 8 feet in length. Gas coming directly from the tank through a short tube may be too cold to use safely in the bowel. A flow of 2 L. per minute is adequate and is of insufficient force to damage mucous membrane. Gas pressure within the bowel cannot be increased so long as the end of the sigmoidoscope is open. The window should never be employed while carbon dioxide is flowing into the bowel.

Explosive gas from higher in the bowel may suddenly be forced into the operative area. To seal off this area Becker[2] and others recommend gentle pressure of the end of the sigmoidoscope against the mucous membrane surrounding the lesion.

CONCLUSIONS

1. Colonic explosions may occur during electrosurgery due to concentration of methane and/or hydrogen.
2. "Purging principles" applied to the colon will prevent gas explosions caused by methane or hydrogen.
3. Carbon dioxide in sufficient concentration will inhibit the explosion of mixtures of hydrogen plus air.
4. The use of a smoke aspirating sigmoidoscope was found to be the most efficient method of introducing carbon dioxide during electrosurgery.

Acknowledgment: The author wishes to express appreciation to C. Eldon White, B.A., Chemical Engineer, for his technical assistance and advice.

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DISCUSSIONS

George H. Thiele (Kansas City, Mo.): Dr. Levy's investigations furnish us with a scientific basis for the prevention of gas explosion. He has outlined in detail a method for utilizing the information which his researches have revealed.

Explosions undoubtedly occur with much

greater frequency than we have been led to believe. A review of the literature reveals that this accident is frequently catastrophic; and when rupture of the colon is widespread, there is at times little that can be done to save the life of the patient in spite of early repair or removal of the ruptured portion of the affected bowel.

Dietary control designed to limit the production of methane and hydrogen, and the use of carbon dioxide in the concentration and with the technic which Dr. Levy has outlined, should do much to afford us all a feeling of security when we are called upon to use electrosurgical methods in the treatment and cure of lesions within reach of the sigmoidoscope.

EDWARD I. LEVY (closing): I wish to thank Dr. Thiele for his kind discussion of my paper. He is correct in stating that dietary changes may also change the hydrogen and methane content of the bowel. Ruge and Lieberman have both commented on this in their papers.

Carcinoma in a Rectal Polyp

REPORT OF A CASE IN INFANCY

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ALTHOUGH the prolapse of a polyp through the anus is not a rare occurrence in childhood, the presence of malignancy in such polyps is seldom suspected. In the case to be described definite malignancy was found in the pedicle near the point of transection, as well as in the distal portion of the polyp.

CASE REPORT

A two year old Mexican child was brought to the hospital due to the presence of a 1½ by 2½ cm. firm, rubbery mass which had prolapsed through the anus two hours previously. The mother stated that the child had passed half a cup of bright red blood since it had appeared. No history of other disease was elicited or any evidence suggesting multiple polyposis in the family. Digital examination revealed the mass to be attached by a short pedicle based about 8 cm. above the dentate line on the left anterior rectal wall. The prolapse was made possible by the mobility of the mucosa in the region of the base.

At surgery a clamp was placed across the base of the pedicle and the growth removed beyond it by sharp dissection. A single layer of atraumatic No. 00 plain catgut suture was used for closure of the 2 cm. incision in the mucosa. After being assured of hemostasis the suture line was allowed to retract to its normal position in the rectum.

When permanent tissue sections were reported to show carcinoma in both pedicle and polyp, a review of the exact site of origin of the polyp was attempted. Encouraged by the report of Smith[1] that the adult sigmoidoscope could be safely used in examining small children, the ¾ inch diameter Boehm sigmoidoscope was introduced and no trace of the pedicle was found. Further examination by this method and by barium enema showed no evidence of the site of removal.

The presence of carcinoma was confirmed by a review of the slides by several pathologists in various parts of the country. (Fig. 1.)

Due to the poor prognosis usually afforded

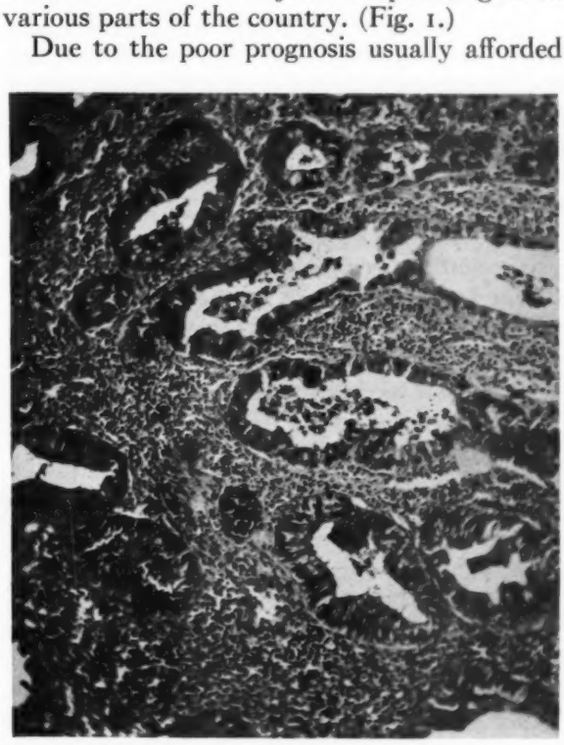

FIG. 1. Carcinoma in a rectal polyp occurring in a two year old child.

patients of this age with carcinoma, regardless of procedure, the majority of surgeons consulted agreed that radical surgery was needed, but that colostomy should be avoided if possible.

The operative procedure devised by Swenson[2] was chosen since it allowed wide excision of the colon above and below the lesion and node-bearing area, and yet resulted in bowel continuity.

The Hiatt[3] modification of the Swenson procedure was carried out and no evidence of spread was found at surgery. Pathologic examination revealed no evidence of carcinoma in the removed colon and rectum.

Following surgery bowel function was restored by the fifth day and was satisfactory thereafter, except for periods of gradual partial obstruction at the suture site at three and six weeks which were relieved by digital dilatation.

Follow-up visits at frequent intervals up to two years after surgery demonstrated no evidence of spread or recurrence. The child was growing and gaining weight normally when last seen.

COMMENTS

The presence of carcinoma is not usually suspected in a solitary polyp prolapsed through the anus of a child, but it should be considered seriously since it has been carefully documented. As listed in Bacon's text,[4] carcinoma of the rectum has been reported in a stillborn monster by Ahfeld[5] and in a three year old child by Lasnier.[6] Oganesyan[7] and others have also reported rectal carcinoma in children, although these were not all polypoid lesions.

The suggestion is made that when this problem is met, an adult sigmoidoscope be passed in order to positively identify the site of origin of the pedicle. Many methods of marking the site of origin of such polyps such as silk suture, skin clips and brain clips have been suggested, but direct vision and measurement from the pectinate line appears to be most practical. The choice of an operative procedure may well be decided by such knowledge, as in this case where a lower origin would have definitely necessitated an abdominoperineal resection of the Miles type.

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Rectal Strictures of Lymphogranuloma Venereum

SOME OBSERVATIONS FROM A FIVE-YEAR STUDY OF TREATMENT WITH THE BROAD SPECTRUM ANTIBIOTICS

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IT has been over five years since Wright and his co-workers' presented an enthusiastic report on the treatment of anorectal lymphogranuloma venereum with chlortetracycline. Subsequently, others reported on the use of chlortetracycline,[2] chloramphenicol,[3,4] oxytetracycline[5,6] and erythromycin[7] in the treatment of this condition. Now after five years it would seem to be of timely interest to give a further report on the effectiveness of the various broad spectrum antibiotics in the management of this infectious disease.

During the past five years chlortetracycline,* chloramphenicol,† oxytetracycline,‡ erythromycin§ and tetracycline‖ have been made available to the Medical College of South Carolina for investigation of anorectal lymphogranuloma venereum. The results obtained during this five-year period form the basis for this report. However, a few general remarks on this disease should precede any discussion of its treatment.

Lymphogranuloma venereum, an infectious disease caused by a virus and transmitted venereally, has many clinical manifestations, of which the anorectal lesions are the most important because they may produce severe structural damage. These lesions are characterized by four factors: (1) chronicity, (2) infectiousness, (3) insidiousness and (4) threat to life. Anorectal lymphogranuloma venereum can and does produce serious complications. At the Medical College of South Carolina cases have been noted of intestinal obstruction due to rectal strictures, of rectums ruptured by bougie dilatations, of epidermoid carcinoma which appeared at the site of rectal strictures and of death from inanition.[8] A casual review of the literature shows that these complications as well as others are far from uncommon.

The initial change in the rectal wall after the onset of the disease is a reddened, friable mucosa which bleeds easily. At this time the patient may notice the presence of blood in the stools. Later a granulation tissue process replaces the mucosa. Bacterial infection and the pyogenic property of the virus cause a purulent anal discharge. At this stage of proctitis the patient may complain of mucus and pus in the stools and sometimes of pruritus ani. The granulomatous process, if untreated, progresses to involve all layers of the rectal wall—mucosa, submucosa, muscularis and tunica—and even the adjacent perirectal tissues. Often there results a more or less complete loss of normal histologic appearance of the rectum and its replacement with a hard, thickened, densely scarred mass. The contraction of the fibrous components of the granulation tissue produces a partial or complete narrowing of the rectal lumen. Partial narrowing of the rectal lumen is termed a stricture; complete narrowing, a stenosis. As the size of the lumen gradually diminishes, increasing difficulty in defecation develops, slowly but progressively. The slowness of the process is typical of the insidiousness of anorectal lymphogranuloma venereum. Also

* The chlortetracycline used was supplied by Lederle Laboratories Division, American Cyanamide Company. The trademark for the antibiotic, chlortetracycline, is aureomycin.®

† The chloramphenicol used was supplied by Parke Davis and Company. The trademark for the antibiotic, chloramphenicol, is chloromycetin.®

‡ The oxytetracycline used was supplied by Chas. Pfizer and Company. The trademark for the antibiotic, oxytetracycline, is terramycin.®

§ The erythromycin used was supplied by Abbott Laboratories. The trademark for the antibiotic, erythromycin, is erythrocin.®

‖ The tetracycline used was supplied by Lederle Laboratories Division, American Cyanamide Company. The trademark for the Lederle brand of tetracycline is achromycin.®

there are cases in which perforations of the bowel wall occur, and such complications as perirectal abscesses, anal fistulas and recto-vaginal fistulas result.

Prior to the advent of the broad spectrum antibiotics the treatment of these rectal strictures was a discouraging problem because it was prolonged, difficult, hazardous and uncertain. The introduction of these antibiotics has changed significantly the management of these cases.

The purpose of this study was to observe both the immediate and the long-term clinical results of the various drugs in cases of anorectal lymphogranuloma venereum. Long-term effects have been observed by recalling the patients who have been treated for examination at periodic intervals. In order to provide for uniformity in systematic investigation special charts were drawn up. They provided for the recording of details of the history, physical examination, laboratory reports and clinical course. Later a proctologic camera for endoscopic views was made available. This camera has been of considerable value in obtaining a lasting objective record of the results for comparative study.

The patients, with few exceptions, came from the surgical clinic of the Medical College of South Carolina. Most were of a low economic group. All except one were Negro women; the one exception was a ten year old Negro boy.[9] All were carefully interviewed with regard to their rectal symptoms. A physical examination was performed in each case with special attention directed toward the anorectal region. Blood counts and urinalyses were also made. All the patients had either a positive virus antigen skin test or a positive complement-fixation test confirming the diagnosis of lymphogranuloma venereum.

Each patient had a rectal biopsy performed before treatment. The histologic sections of such biopsy specimens showed only chronic inflammation. This biopsy was considered to be of paramount importance in ruling out malignancy. In previous experiences at the Medical College surgical clinic it had been noted that carcinoma had developed in cases of rectal strictures. Rectal biopsies were taken both before and after treatment in order to have objective evidence of the effectiveness of the drugs, but the biopsies taken after treatment were discontinued because of the relatively meager definitive information obtained and also because of their adverse psychologic effect upon the patients.

While the methods used in this study are admittedly relatively crude, they are practical and reasonable. The reliability of an uneducated patient's history is always open to question. However, it would seem that a patient previously treated with various medications and rectal dilatations over a period of years should be able to give some opinion of the value of the new drug used. The difficulty of accurately measuring a rectal stricture and its response to treatment is admitted frankly. In order to evaluate the effectiveness of therapy a system of classification and grading of rectal strictures was instituted. This system of classification is incorporated in the special chart used to record the data in this investigational project.

During the past five years twelve cases of rectal strictures of lymphogranuloma venereum were treated with chlortetracycline, eight with chloramphenicol, eight with oxytetracycline, three with erythromycin and three with tetracycline. The results of treatment, presented in Tables I to V, were evaluated on the basis of periodic follow-up studies. The longest follow-up studies, now covering nearly five years, are those of the chlortetracycline series.

Some general impressions have been formed over the years after treatment and follow-up of these cases of rectal strictures. The action of the antibiotics is directed toward the inflammatory component of the rectal stricture. The drugs have no known effect on scar tissue. As demonstrated by biopsy study an inflammatory component of greater or less degree is present in most, if not all, rectal strictures. By reducing the inflammatory edema of the rectal stricture, the drug produces an increase in the size of the rectal lumen. In early rectal strictures with relatively greater inflammatory and lesser cicatricial components the drugs may be expected to produce good results. In old rectal strictures with relatively less inflammatory and greater cicatricial components the drugs may be expected to be less effective.

Usually the patient receiving the drug reported a feeling of improvement within three to five days. Improvement of varying degrees was reported by all. Objective evidence of improvement was observed in most cases.

Chlortetracycline, chloramphenicol, oxytet-

racycline and tetracycline in dosages of 750 to 1,000 mg. daily for twelve to twenty-five days have been sufficient to produce a satisfactory clinical improvement. Erythromycin in dosages of 1,600 mg. daily for a similar duration was effective. By comparison, however, in the limited number of cases studied, erythromycin was less effective than chlortetracycline, chloramphenicol and oxytetracycline. Tetracycline has been used for too short a period of time to be evaluated adequately at this time.

Since there are no laboratory criteria for determining the adequacy of treatment of the individual case, the following clinical criteria have been evolved and employed at this clinic.

TABLE 1
CHLORTETRACYCLINE CASE SUMMARIES

Patient and Dose	Symptoms	Stricture*	Results
Case 1. A. B. 250 mg. q.i.d. × 12	Rectal bleeding; mucus per rectum; pain on defecation	Tubular; type 3; grade 3	Good 25 mo.; no bleeding; no pain with bowel movements
Case 2. A. H. M. 250 mg. q.i.d. × 12½	Pencil-sized stools; straining at stool; painful defecation	Tubular; type 4; grade 4	Good 40 mo.; no straining or pain; larger caliber stools
Case 3. J. M. B. 250 mg. q.i.d. × 12½	Rectal bleeding; daily laxative; painful defecation	Annular; type 2; grade 2	Equivocal 27 mo.; no bleeding; laxative q. 2 wk.
Case 4. W. S. L. 250 mg. q.i.d. × 12½	Constipation; rectal bleeding; pain on defecation	Annular; type 2; grade 2	Good 56 mo.; no pain with bowel movements; no laxative since drug
Case 5. W. M. 250 mg. t.i.d. × 5 250 mg. b.i.d. × 5	Rectal bleeding; straining at stools; narrowed stools	Annular; type 3; grade 4	Good 54 mo.; one episode slight bleeding in Aug. 1952
Case 6. E. M. H. 250 mg. t.i.d. × 5 250 mg. b.i.d. × 4	Pencil-sized stools; painful defecation; rectal bleeding	Annular; type 3; grade 3	Good 40 mo.; no rectal bleeding; no pain with bowel movement
Case 7. C. E. 250 mg. t.i.d. × 6 250 mg. b.i.d. × 2	Rectal bleeding; narrowed stools; painful defecation	Annular; type 3; grade 3	Good 35 mo.; no rectal bleeding; no pain with bowel movement
Case 8. J. R. R. 250 mg. t.i.d. × 5 250 mg. b.i.d. × 5	Rectal bleeding; mucus from rectum; straining at stool	Tubular; type 4; grade 3	Good 53 mo.; no bleeding or mucus; no straining
Case 9. R. W. 250 mg. t.i.d. 13 16 months later 250 mg. t.i.d. × 14	Rectal bleeding; constipation; painful defecation	Annular; type 2; grade 3	Good 26 mo.; no bleeding; less pain with bowel movement
Case 10. V. L. 250 mg. t.i.d. × 5	No bleeding; no mucus; no pain with bowel movement	Tubular; type 4; grade 3	Equivocal 45 mo.; "felt improved" after drug; no pain or bleeding
Case 11. H. H. 250 mg. t.i.d. × 5	Pain with bowel movement; no blood	Tubular; type 4; grade 3	Equivocal 43 mo.; less pain with bowel movement; no bleeding
Case 12. L. S. 250 mg. t.i.d. × 10 23 months later 250 mg. t.i.d. 14	Straining with bowel movement; rectal bleeding	Tubular; type 4; grade 3	Good 41 mo.; less straining; rectal bleeding once

* Explanation for classification and grading: *Type:* Annular or tubular, singular or multiple (over 1.5 cm. in length is to be considered tubular). Type 1—soft, smooth and regular due to mucosal edema and congestion; type 2—hard and irregular with granulomatous deposits in the mucosa and submucosa; type 3—small amount of scar tissue in the submucosa and muscularis; type 4—dense scar tissue in all layers or rectum and perirectal tissues. Grading according to size of diameter: grade 1, 1½ inches (3.7 cm.); grade 2, 1 inch (2.5 cm.); grade 3, ½ inch (1.2 cm.); grade 4, ¼ inch (0.6 cm.); grade 5, admits none.

Patient's observations: (1) absence of rectal bleeding; (2) diminution of straining at stool; (3) reduction of pain at defecation and (4) absence of mucus and purulent exudate from

TABLE II

CHLORAMPHENICOL CASE SUMMARIES

Patient and Dose	Symptoms	Stricture*	Results
Case 1. J. D. 250 mg. q.i.d. × 21 6 months later 250 mg. t.i.d. × 7	Rectal bleeding; narrowed stools; mucus per rectum	Annular; type 4; grade 4	Good 38 mo.; bowel movements easier; no bleeding; stools larger in size
Case 2. S. G. 250 mg. q.i.d. × 15	Straining with defecation; rectal bleeding	Annular; type 3; grade 4	Good 6 mo.; no mucus or bleeding; no straining
Case 3. A. S. 250 mg. q.i.d. × 8	Laxative 2–3 × wk.; straining at stool	Annular; type 3; grade 3	Good 39 mo.; "seems" to have helped; stools larger
Case 4. M. G. 250 mg. t.i.d. × 26	Rectal bleeding; pain with defecation	Annular; type 3; grade 4	Good 12 mo.; no pain with bowel movement; no bleeding
Case 5. E. H. 250 mg. q.i.d. × 7 250 mg. t.i.d. × 7	Straining at stool; feces per vagina since Nov. 1950; colostomy	Tubular; type 4; grade 4; rectovaginal fistula demonstrable	No improvement 14 mo.; following colostomy and after 2 wk. of drug rectovaginal fistula was not demonstrable; gradual and progressive obliteration of lumen
Case 6. J. G. 250 mg. t.i.d. × 35 250 mg. q.i.d. × 7 250 mg. t.i.d. × 9	Frequent bowel movements; narrowed stools; straining at stools; rectal bleeding	Annular; type 3; grade 4	Good 38 mo.; no bleeding; no straining at stool; stools larger
Case 7. M. F. 250 mg. t.i.d. × 28	Pain with defecation; rectal bleeding; pruritus ani	Tubular; type 3; grade 2; multiple fistulas in-ano	Equivocal 29 mo.; no bleeding; fistulas-in-ano persisting
Case 8. E. W. 250 mg. t.i.d. 14	Straining at stool; occasional laxative	Tubular; type 4; grade 3	Good 30 mo.; no laxative; no straining

* See explanation under Table 1.

TABLE III

OXYTETRACYCLINE CASE SUMMARIES

Patient and Dose	Symptoms	Stricture*	Results
Case 1. B. H. 250 mg. t.i.d. × 16 250 mg. t.i.d. × 7	Rectal bleeding; pain on defecation; laxative 2–3 × wk.	Tubular; type 4; grade 4	Good 30 mo.; no bleeding or pain; no laxative
Case 2. C. B. 250 mg. t.i.d. × 20	Laxative 2–3 × wk.; straining at stool	Annular; type 3; grade 3	Good 27 mo.; seldom uses laxative; no straining
Case 3. E. W. 250 mg. q.i.d. × 14	Rectal bleeding; pain on defecation; straining at stool	Tubular; type 4; grade 4	Good 26 mo.; no bleeding; no straining with bowel movement; no pain with bowel movement
Case 4. L. G. 250 mg. q.i.d. × 25	Laxative 2–3 × wk.; rectal bleeding; narrow stools	Tubular; type 2; grade 4	Good 23 mo.; stools larger; no bleeding; no laxative
Case 5. L. W. 250 mg. q.i.d. × 25	Narrowed stools; rectal bleeding; needs laxative	Tubular; type 4; grade 4; rectovaginal fistula	No improvement 23 mo.; infrequent rectal bleeding; recurrent drainage from rectovaginal fistula
Case 6. M. J. J. 250 mg. q.i.d. × 25	Rectal bleeding; ribbon stools; mucus per rectum	Annular; type 2; grade 3	Good 19 mo.; no rectal bleeding; no mucus; increase in size of stools
Case 7. J. C. 250 mg. q.i.d. × 14	Ribbon stools; pain on defecation	Annular; type 2; grade 4	Good 5 mo.; stools larger; no pain on defecation
Case 8. I. R. 250 mg. q.i.d. × 14	Pain on defecation; rectal bleeding	Annular; type 3; grade 4	Good 3 mo.; no pain on defecation; no rectal bleeding

* See explanation under Table 1.

the rectum. *Physician's observations:* (1) rectal wall and stricture changed to lining of smooth, gray appearance and (2) apparent evidence of larger lumen of rectal stricture.

These criteria also were used to determine the need for retreatment. There has been no

TABLE IV

ERYTHROMYCIN CASE SUMMARIES

Patient and Dose	Symptoms	Stricture*	Results
Case 1. E. T. 200 mg. q.i.d. × 12½	Rectal bleeding; narrowed stools	Annular; type 3; grade 4	Good 14 mo.; no rectal bleeding; larger caliber stools
Case 2. P. W. 200 mg. q.i.d. × 25	Rectal bleeding	Tubular; type 3; grade 3	Good 12 mo.; no rectal bleeding
Case 3. L. W. 200 mg. q.i.d. × 17 (previous treatment with oxytetracycline)	Rectal bleeding; rectovaginal fistula; mucus per rectum	Tubular; type 4; grade 4; rectovaginal fistula	No improvement 12 mo.; recurrent drainage from rectovaginal fistula

* See explanation under Table 1.

TABLE V

TETRACYCLINE CASE SUMMARIES

Patient and Dose	Symptoms	Stricture*	Results
Case 1. A. M. 250 mg. q.i.d. × 14	Narrow stools; constipation; straining at stool	Tubular; type 3; grade 3	Good 6 mo.; stools larger; no straining at stool; daily bowel movement
Case 2. K. B. 250 mg. q.i.d. × 15	Narrow stools; pain on defecation	Tubular; type 3; grade 4	Good 6 mo.; stools larger; no pain on defecation
Case 3. G. R. G. 250 mg. q.i.d. × 14	Constipation; narrow stools; pain on defecation	Tubular; type 3; grade 3	Good 2 mo.; stools larger; no pain on defecation

* See explanation under Table 1.

reluctance to retreat these patients with recurrent symptoms from rectal strictures of lymphogranuloma venereum.

Digital and bougie dilatations were not used in the treatment of the strictures. In addition to the danger of perforations and its sequelae, the dilatations may tend to produce more inflammation and more scarring.

Serial quantitative complement-fixation tests for lymphogranuloma venereum have been employed in this study. The blood was tested before treatment and whenever the patient returned for a follow-up visit. The periodic complement-fixation reactions have been employed to measure the effect of the antibiotics on the antibody titer of lymphogranuloma venereum. These quantitative complement-fixation studies, made in the Bacteriology Department of the Medical College, are still in progress and will be reported in greater detail separately. Preliminary observations, however, indicate that the antibiotics have no immediate influence on the antibody reaction of the virus of lymphogranuloma venereum. The use of serial quantitative complement-fixation tests seems to be of limited practical value in the management of anorectal lymphogranuloma venereum.

From a practical aspect, most patients apparently are able to have satisfactory bowel movements if the lumen permits the tip of the finger to pass. Here it is important to know whether one is dealing with an annular or a tubular stricture. In a tubular stricture the diameter of the proximal portion is an unknown factor (unless roentgenograms are employed). It is important to remember that the antibiotics will not replace surgery in certain cases. At this clinic no case of fistula-in-ano or of

rectovaginal fistula has been cured by any of these drugs alone.

SUMMARY

Observations have been made on the use during the past five years of the broad spectrum antibiotics in the treatment of rectal strictures due to lymphogranuloma venereum.

Because there is an inflammatory component in most, if not all, such rectal strictures, the use of one of the broad spectrum antibiotics is indicated.

At this clinic improvement of varying degree was reported by all patients. Objective evidence of improvement was observed in most cases.

Serial quantitative complement-fixation tests for lymphogranuloma venereum do not appear to be reliable indices for determining adequacy of treatment or of the need for retreatment.

Since the decision to continue treatment with the antibiotics or to retreat these cases of rectal strictures of lymphogranuloma venereum must be made solely on clinical evidence, there is a definite need for clinical criteria for determining adequacy of treatment. Such criteria evolved at this clinic during the past five years have been presented.

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DISCUSSION

Robert S. Smith (Washington, D. C.): I would like to compliment Dr. Banov on this excellent paper. Most of us do not have personal experience with a sufficient number of cases of lymphogranuloma venereum to offer valid criticism. In Washington, D. C. most of the cases are cared for by the Bureau of Venereal Diseases of the Health Department. The records of several of the general hospitals reveal that only a very occasional case is filed under the diagnosis of lymphogranuloma venereum.

Upon investigating the incidence of the condition in the V. D. Clinics, I learned some very interesting facts, which I should like to relate, regarding the over-all picture of this disorder. Dr. Aston B. Greaves, of the District of Columbia Health Department, has spent most of his time and energy for a number of years on this very problem. He has been assisted by a number of advantages which are probably not generally available. In the first place, the V. D. Clinics supplied a large number of potential cases of this disease. Secondly, he and the laboratory personnel of the Health Department have been able to have the intimate cooperation of Drs. Nigg, Hilleman and Bowser of the Army Medical Service at Walter Reed Hospital.

These later investigators have increased the sensitivity of the compliment-fixation test many times by using a phenol-enhanced yolk sac antigen. Their modified procedure should prove to be a useful laboratory criterium for early detection and a quantitative evaluation of the progress of this disease.

The compliment-fixation test was carried out on 2,029 new patients at the Polk V. D. Clinic of the District of Columbia from January 1 to March 22, 1954. Of these, 1,515 were negative and 514 were positive (25.3 per cent). A Frei test was done on 291 of the positive-reacting patients. Two hundred twenty-three of these could not be followed. Of the 291 positive compliment-fixation cases, 159 were negative to the Frei test and 132 were positive (45.4 per cent). Of the 514 patients with positive compliment-fixation tests, 494 were asymptomatic (43 per cent of these were Frei positive); 13 had minor or early symptoms (90 per cent

of these were Frei positive), and 7 revealed late symptoms (71 per cent of these were Frei positive).

These data indicate that the incidence of early or "silent" cases of lymphgranuloma venereum, detected by serologic methods, is considerable and out of proportion to the relatively small number of advanced or clinically obvious cases presenting anorectal symptoms. This discrepancy may be due to a number of factors: (1) Patients may be carriers with a high resistance; (2) there may be spontaneous cures, for in certain instances the disease may be self limited and (3) cures may have resulted from the application of antibiotics coincidentally for other illnesses, as for example, sore throats, other V. D. infections, etc. It appears justified to conclude that the cases reported by Dr. Banov are patients with little intrinsic resistance to the disease and his results are thus all the more significant.

Treatment of these early cases by sulfa drugs and several antibiotics is now underway. Observation of a control group without therapy is being attempted. This is difficult for at this time it is not easy to insure that these patients do not receive any antibiotic medications for coincident illnesses. It is too early to report results, but continuing studies of the serial titer of the compliment-fixation tests are being conducted. It is hoped that by these means early cases may be treated before the development of serious lesions and thus the disastrous consequences of this progressive disorder *prevented.* I believe that the chemotherapeutic agents have a greater effect on the early cases than on the late cases.

I am sure we will all be interested in seeing the future reports of Dr. Aston Greaves and his co-workers, as we will be in further reports from Dr. Banov. There is still much work to be done.

Anorectal Fistulas

AN ANATOMIC CLASSIFICATION AND PRINCIPLES OF TREATMENT

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THE purpose of this paper on anorectal fistulas is to present a concise classification and also the general principles to be considered in the surgical treatment of these fistulas. The pathogenesis is not to be discussed other that to limit the scope only to those fistulas resulting from an infectious process, regardless of whether it originates intra- or extra-anally or intra- or extrarectally. The anatomic location of the original abscess and the establishment of the main track determines not only the proper classification but also the method of eventual surgical approach.

It is true that every fistula of inflammatory origin is preceded by an abscess and the main track of the fistula is the contracted but unobliterated original abscess cavity. Therefore, the anatomic site of the original abscess determines the type of fistula. With this in mind the following classification of anorectal fistulas appears fit and proper from an anatomic standpoint:

I. Subcutaneous Anorectal Fistula:
- A. Incomplete external
- B. Incomplete internal
- C. Complete

II. Submucous Anorectal Fistula:
- A. Incomplete external
- B. Incomplete internal
- C. Complete
- D. Bilateral

III. Intermuscular Anorectal Fistula:
- A. Incomplete internal

IV. Subsphincterio Anorectal Fistula:
- A. Incomplete internal
- B. Complete
- C. Bilateral or anterior horseshoe

V. Ischiorectal Fistula:
- A. Incomplete external
- B. Incomplete internal
- C. Complete
- D. Bilateral or posterior horseshoe

VI. Pararectal Fistula:
- A. Incomplete internal
- B. Complete
- C. Bilateral

SUBCUTANEOUS TYPE OF ANORECTAL FISTULA

The subcutaneous type of anorectal fistula is found only in the subcutaneous tissue close to the anus. The external opening, as a rule, is small and contracted and is usually situated within an inch of the anal verge. The main track with any connecting track or tracks may usually be found superficial to the deep fascia and the external sphincteric musculature. This type is a so-called simple type, is never extensive and is usually unilateral.

The incomplete external subcutaneous fistula may have a very small pinpoint opening with the main track directed to the anal margin. This type may be found anywhere around the anus. It can be palpated very readily and when probed the track is usually very short, being not more than ½ inch as a rule.

The incomplete internal subcutaneous fistula is almost always associated with an anal fissure or an anal ulcer. The opening is found at the distal portion of the fissure or the ulcer and the main track extends in a straight line from the anus in a radial manner. This type predominates either anterior or posterior because of the association with fissure or ulcer.

The complete subcutaneous fistula is usually a further stage of the above type with the external opening occurring within an inch from the anal verge. Because the internal opening enlarges as the fissure becomes an ulcer, we find fecal matter entering it and setting up a constant suppurative process. In this way offshoots may form and terminate in many external openings.

SUBMUCOUS TYPE OF ANORECTAL FISTULAS

The submucous anorectal fistula is limited to the submucosa of the lower part of the rectum and may be unilateral or extend to both sides of the rectum. There may be multiple submucosal fistulas also, each fistula being distinctly apart from the other. It may extend laterally as well as downward to the anus where the external opening may be at the anorectal margin or extend under the anoderm

and discharge within ½ inch of that margin out through the skin. It may be found also in conjunction with a subcutaneous fistula where the external opening is found at the proximal edge of an anal fissure or anal ulcer.

The incomplete external submucosal fistula results from an abscess of the submucosa of the lower rectum wherein the pus migrates toward the anus and discharges within one-half of the anal verge. The main track runs usually in an oblique direction ending cephalad in a blind pouch.

The incomplete internal submucous fistula is the type usually found in conjunction with a subcutaneous fistula or as a complication of an anal fissure or an anal ulcer. The opening is in the proximal end of the fissure or ulcer and is considered internal because the opening is at the anorectal junction. The main track extends upward next to the muscular coat and beneath the hemorrhoidal plexus. It is seldom more than 1 inch long. This type may also be found as part of an ischiorectal fistula.

The complete submucous fistula, although uncommon, exhibits an internal opening above the attachments of the levator musculature with the track extending toward the anus and discharging either at the anorectal junction or through the skin within ½ inch of the anal verge.

The bilateral submucous fistula is really a further complication of the incomplete submucous fistula in which the pus extends transversely in the posterior submucosa and eventually discharges at the anorectal junction in the midline posteriorly. This results in a T-shaped track due to the contraction of the abscess cavity following evacuation of the pus through the opening at the anorectal junction.

INTERMUSCULAR TYPE OF ANORECTAL FISTULA

The intermuscular type of anorectal fistula is one in which the main track runs between the internal sphincter and the longitudinal muscular coat of the rectum. It is similar to the incomplete submucous internal fistula, the only difference being that it is located between the two muscular coats of the rectum instead of between the mucosa and the internal sphincter. It is usually arranged longitudinally with the wall of the rectum and is never more than 1 inch long. The internal opening is found at the anorectal junction, and the track extends cephalad and ends in a blind sac. It is one that is seldom seen but must be reckoned with. The surgery of this type of fistula is to lay it open from the internal opening to the culmination of the sac. The division of the internal sphincter in no way interferes with control. One must be careful, however, not to extend the incision through the longitudinal muscular layer of the rectum.

SUBSPHINCTERIC TYPE OF ANORECTAL FISTULA

The subsphincteric type of anorectal fistula is the most common type of fistula which is associated anatomically with the anorectal musculature. Here we find the main track ending under the subcutaneous external sphincter and any internal opening is found at the intermuscular septum where some fibers of the longitudinal muscular coat of the rectum terminate in the anal canal.

The incomplete internal subsphincteric fistula and the complete subsphincteric fistula are really stages of the same type. We see it in the primary abscess stage with the abscess situated beneath the external sphincter and the pus migrating to the outer border of that muscle. When the opening occurs spontaneously, it usually discharges through the skin about 1½ inches from the anal verge. This constitutes the second type or complete subsphincteric fistula. There may be subcutaneous extensions of the main track with secondary external openings. When the primary external opening is situated anteriorly to the transverse anal line, the offshoots extend forward and toward the midline; but when the primary external opening is situated posteriorly to the transverse anal line, the offshoots extend backward and outward. Here it is that Goodsall's law applies as to the location of the internal opening. When the primary external opening is situated anteriorly to the transverse anal line or on that line, the internal opening is directly opposite to it in the same radial line, between the sphincters and the track is in a straight line. When the primary external opening is situated posteriorly to the transverse anal line the internal opening will be found in midline posteriorly between the sphincters with the track curving backward and inward. This makes it difficult to pass a probe from the external opening through the main track out through the internal opening.

The bilateral subsphincteric fistule constitutes what is often called an anterior horse-

shoe fistula. This is the result of subcutaneous offshoots extending to both sides of the midline. Where the primary external opening is anterior and close to midline, all other characteristics of the subsphincteric fistula are present.

ISCHIORECTAL TYPE OF ANORECTAL FISTULA

The ischiorectal type of anorectal fistula occurs in less than 30 per cent of the total number of anorectal fistulas. Here the abscess occurs in the ischiorectal fossa and so the main track is confined to the ischiorectal fossa. The internal opening is always found in the posterior commissure of the anus in the midline between the sphincters. It is interesting to note that, as described by Miles, there is always a separation between the sphincters especially pronounced in the posterior midline of the anal canal. The external opening is generally situated about 1½ inches from the anal verge and slightly behind the transverse anal line. Thus the main track is curved backward from the external opening to the internal opening. The infection may burrow through the posterior communicating space to the opposite fossa and result in a bilateral ischiorectal abscess with one internal opening and several external openings. This is the so-called posterior horseshoe fistula.

The incomplete external ischiorectal fistula is unique in that it is usually caused by trauma with resulting necrosis of the ischiorectal fat and eventual discharge of the necrotic material through an external opening situated anywhere in the perianal area. There is no internal opening although the abscess may extend to the intersphincteric line in the posterior commissure but not penetrating into the rectum.

The incomplete internal ischiorectal fistula is usually the preliminary stage of the complete ischio rectal fistula. The internal opening is found in the midline posteriorly as previously described and results in an abscess which later becomes a complete fistula either by spontaneous rupture externally or by surgical incision and drainage of the abscess.

The bilateral ischiorectal fistula is the extension of the original focus to the opposite side. There is always one internal opening in the usual posterior midline location with both ischiorectal fossa involved as the main tracks. One may find a complete ischiorectal fistula on one side and an incomplete internal ischiorectal fistula on the other. In rare cases, usually of long-standing infection and recurrent abscesses, a deep offshoot may extend into the anterior ischiorectal fossa and evacuate pus through an external opening in front of the anus close to the midline. Very occasionally one sees the anus completely surrounded by a fistulous track with several secondary external openings on both sides but always only one internal opening present which is situated in the midline, posteriorly between the sphincters.

PARARECTAL TYPE OF ANORECTAL FISTULA

The pararectal type of anorectal fistula is the result of an abscess of the pararectal space between the wall of the rectum and the fascia propria. This dense fascia will wall it off from the loose cellular tissue between the pelvic peritoneum, the levator and or the supralevator space. The pus then subsequently may surround the rectum or invade the subadjacent ischiorectal fossa and create an ischiorectal abscess with its subsequent termination.

The incomplete pararectal fistula is usually the first stage in the development of a complete pararectal fistula. Here early diagnosis and the proper surgical technic will prevent much suffering. However, many times the extension to the ischiorectal fossa is the first definite objective symptom although the patient has had deep seated symptoms go unrecognized.

GENERAL PRINCIPLES OF SURGICAL TREATMENT

The operative technic for most anorectal fistulas is rather simple because the greater percentage of fistulas is of the uncomplicated type. This necessitates opening the track from the external to the internal opening by dividing the tissues over it. A thin, supple wire probe is introduced into the external opening, extended through the main track and brought out through the internal opening. A No. 1 lacrymal probe is very helpful, especially when the track is contracted. The popular stiff, thick probe has no place in surgery of anorectal fistulas. The tissues over the probe are then incised until the probe is free. In the subcutaneous fistulas these tissues consist of only skin, subcutaneous tissue and probably fibers of the corrugator cutis ani muscle. Of course when a fissure or ulcer is associated with the fistula, a posterior proctotomy should be done also, severing the fibers of the subcutaneous external sphincter. An adequate external incision

should be made to allow for proper drainage. Only a small pledget of oxydized cotton may be used as a dressing after all hemostasis has been secured.

When operating upon a submucous fistula one must be certain that the abscess or main track is confined to the submucous layer and does not extend through the rectal wall and continue external to it. A probe inserted into a submucous fistula can be readily felt under the mucosa by the examining finger in the rectum. The surgical treatment consists of laying open the rectal mucosa overlying the probe. If the probe cannot be easily palpated by the examining finger in the rectum, one can be assured the track is pararectal and division of the overlying tissues would certainly impair sphincter control. Consequently one must be sure of his diagnosis of a submucous abscess or fistula and be certain not to extend the incision through the wall of the rectum but only down to it. Profuse bleeding may be encountered when the track extends under a hemorrhoidal pedicle and necessitates complete hemostasis. This demands a good exposure, a good light and the meticulous catching of each bleeder and tying it off separately.

Operative treatment of the subsphincteric fistula consists of laying open the tissues above a probe extending through the main track proceeding from the external opening toward the internal opening. In the anterior fistula the main track is straight and the probe proceeds from the external opening through the main track to the internal opening and into the anus. All offshoots should be carefully sought after and exposed. In the anterior horseshoe fistula this sometimes means a very extensive excision, but must be done to cure the fistula.

In the posterior horseshoe fistula similar action must be taken—the fistula followed from the primary external opening to the internal opening. This often leads to an extensive dissection and allows the anus to retract. Whenever an anterior horseshoe fistula and a posterior horseshoe fistula is found in the same patient, I believe a stage operation should be performed, doing one fistula and allowing the tissues to heal and then completing the operation in a second stage. I always like to do the fistula that is most complicated first and it is usually the posterior type. Here it is that the "relieving cuts" described by Miles are very necessary. If one tries to saucerize an extensive fistula, so much skin and subcutaneous tissue is sacrificed that the resulting scar distorts the anus very much. The principle of the "relieving cuts" is that tension is relieved in the direction of muscular pull so that the wound gapes but lymphatics are not embarrassed. Plastic surgeons utilize this principle in their work. Even though our cosmetic results are not important, we must have good functional results. Finally, in this type of fistula we should always carefully search for a submucous extension or an intermuscular complication originating from the internal opening.

SURGERY OF ISCHIORECTAL FISTULA

In treating an ischiorectal fistula the same principles are utilized. If the fistula is an incomplete external type and extends between the sphincters and to the mucous membranes, it is good judgment to treat it as a complete fistula and separate the muscle and membrane.

In a complete ischiorectal fistula the probe should be passed gently and the pyogenic membrane of the track identified. Also any offshoots should be opened. I find it very messy to use any dye to identify the track and the identification of pathologic tissue is made much more difficult. If the track extends anteriorly beneath the inferior hemorrhoidal vessels, it should not be laid open as this will destroy the nerve supply to two-thirds the external sphincter and a consequent postoperative seepage may occur. The part behind and in front of the vessels may be opened and exposed but the vessels left intact. Careful postoperative care will allow the deep portion of the underlying fistula to heal in.

PARARECTAL FISTULA

The procedure in opening an incomplete pararectal fistula, after inserting a finger into the rectum and palpating the abscess, should be to insert a pointed scalpel through the perianal skin lateral to the anus and move it cephalad, parallel to the rectal wall until the abscess is reached and pus exudes through the external incision. Care should be taken not to penetrate the wall of the rectum but continue the incision down to the internal opening. A finger is inserted into the abscess cavity to determine if any foreign body is present, a common cause of a pararectal abscess. Adequate drainage is then established as described. All incisions should be large enough to insure

perfect drainage yet not sacrifice too much skin and subcutaneous tissue. All abscesses of the pelvirectal and retrorectal space should be opened in a similar manner. An extra rectal abscess should never be opened from the inside of the rectum.

POSTOPERATIVE TREATMENT

The after treatment of anorectal fistula must be careful and meticulous. The object is to have the postoperative wound heal progressively from its deepest part throughout its entire extent. Daily dressing and close observation are necessary. Adequate drainage must be maintained. In the supralevator fistulas this means keeping the drainage exit through the levator ani muscle open until the cavity above the levator is closed. Allowing a pack to remain in the cavity two to four days with segmental removal each day, keeps the walls from falling together too quickly. Once the pack is removed it is never repacked. The granulation tissue will be kept under control by curetting any excess. All bridging or shelving must be prevented and the drainage exit kept open by subsequent reincision if necessary. The use of a potassium permanganate solution in about a 1:8,000 strength as an irrigation after bowel actions is also beneficial. Should pus collect in the wound, it is evident that active suppuration is taking place somewhere in the healing space and search must be made for undiscovered offshoots or some bridging or shelving.

SUMMARY

A classification of anorectal fistulas resulting from infectious processes has been presented. This classification is based on the anatomy of the anorectal region and the location of the original abscess. The general principles of surgical treatment and after care of anorectal fistulas have also been reviewed.

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A Reevaluation of the Factitial Proctitis Problem

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IT is evident that cure rates for carcinoma of the uterine cervix are higher than ever before in the hands of those experienced with irradiation therapy of these lesions.[1] To achieve these encouraging results increasing dosages of irradiation are being administered. More irradiation is being given the League of Nations Stage III lesion, and even palliation of the Stage IV lesions is being attempted. As a result, proctologists must be willing to accept the increased incidence of factitial proctitis.

It is a recognized and accepted fact that unless deep therapy is administered in the maximum dosage which will be tolerated, maximum possible cure rates will not be obtained. Until a method is devised to provide complete shielding of the organs adjacent to the uterus, some degree of secondary irradiation damage of these structures must be anticipated. The rectum, by virtue of its position close to the uterus, is one of the organs most frequently involved with the secondary effects of irradiation. With the standard technic used at the University of Minnesota approximately 7,000 gamma roentgens are given to the anterior rectal wall.

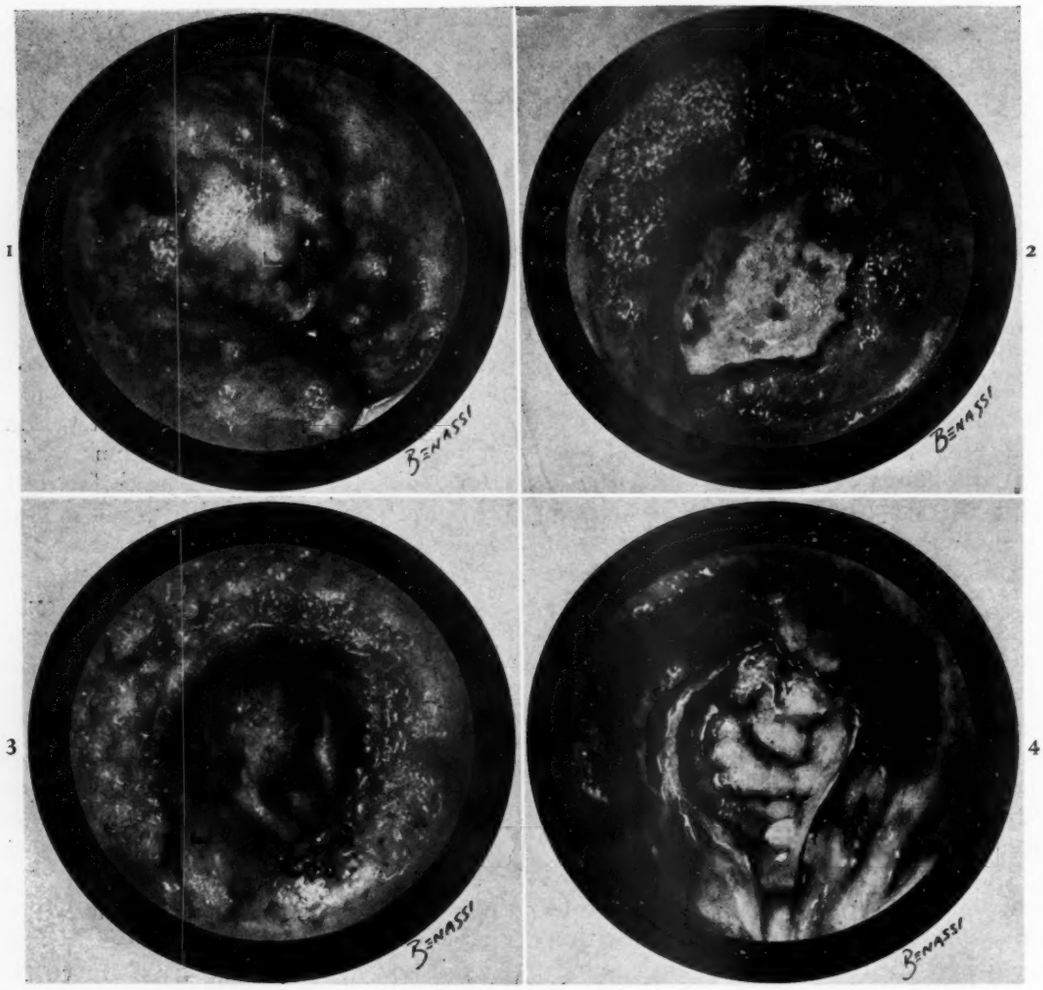
Proctologists, as well as gynecologists, are aware that the medical and surgical therapy available for patients with factitial proctitis has been inadequate. In fact, no specific plan of therapy has been proposed except for local hygiene and performing colostomies upon those patients with strictures and fistulas. It is the purpose of this paper to propose a more efficacious regimen of treatment, particularly for those patients without gross bowel deformities.

This represents a preliminary report of those patients with factitial proctitis seen in the Proctology Clinic at the University of Minnesota Hospitals during the past year. All patients receiving irradiation therapy during the past year for carcinoma of the uterine cervix were sigmoidoscoped before treatment and at three-month intervals postirradiation. These patients were instructed to report to the Proctology Clinic sooner if symptoms of proctitis developed. Those patients with factitial proctitis were followed closely at two- to four-week intervals. Again it must be stressed that this is a preliminary report with emphasis on therapy.

History and Incidence. Gastrointestinal injury following irradiation was first described by Walsh[2] in 1897. He cited a case of a man in whom diarrhea developed after several weeks of exposure to x-rays. During a two-month vacation the symptoms subsided, only to recur on subsequent exposure. In 1930 Buie and Malmgren[3] were the first to apply the term "factitial proctitis" to irradiation injury of the rectum. Since that time numerous authors have covered all aspects of the problem in detail and most of them have agreed that the process is self limited after the lapse of considerable time.

There is wide variation in the reported incidence of factitial proctitis in the literature. Buie and Malmgren reported that this lesion occurred in only 3 per cent of the irradiated patients in their original paper. Todd[4] reviewed the incidence as reported by several clinics and found that approximately 5 per cent of the patients had severe rectal changes postirradiation. In 1942 Aldridge[5] reported that 16.9 per cent of his patients developed some degree of intestinal injury. It was his opinion that most reports heretofore included only those patients with the more serious injuries with symptoms requiring active treatment or surgical intervention.

In order to estimate accurately the correct incidence of factitial proctitis, all patients receiving irradiation should be followed periodically for at least a five-year period. Since the patients in our series have only been



FIGS. 1 to 4.

followed for a year, no attempt will be made to compute the incidence of factitial proctitis postirradiation with this group. It is the author's belief that with the increased dosages of x-ray, cobalt and radium that are being advocated, along with the closer follow-ups, the incidence could be higher than that reported by Aldridge.

Classification of Proctitis. The various degrees of proctitis are classified into four grades in order to promote a better understanding between the departments at the University of Minnesota Hospitals as to the degree of involvement and secondly, to differentiate those lesions that may respond to medical treatment from those that merit surgical attention. The degrees of tissue damage as observed in these patients fall into four categories rather naturally. The classification is made entirely on the proctoscopic appearance of the bowel. Grade I: (a) Localized erythema and telangiectasis; friable mucosa that bleeds easily; no ulceration or stricture (Fig. 1.); (b) erythema is more diffuse with accompanying periproctitis, marked pain and sensitivity. Grade II: Characterized by the presence of ulceration with a greyish tenacious slough usually involving the anterior rectal wall; proctitis as seen with Grade I lesions usually accompanies the ulceration, except in the late stages of the disease. (Fig. 2.) Grade III: Characterized by the presence of a stricture, plus proctitis and ulceration; either of the latter may coexist in varying degrees. (Fig. 3.) Grade IV: Characterized by proctitis, ulceration, stricture and a rectovaginal fistula or bowel perforation. (Fig. 4.)

Clinical Features. The symptoms of irradiation injury to the rectum or distal sigmoid colon may appear within the first month following irradiation, or as late as seven years after-

ward.[6] Six months is the average time interval for symptoms to develop. The symptoms depend entirely upon the degree of proctitis and the stage of its development. Bleeding and the passage of mucus is usually the first sign noticed by the patient. This may be accompanied by diarrhea and misinterpreted as "colitis." These may be the only symptoms from a Grade I lesion. A number of patients have been observed proctoscopically with Grade I lesions, mucosal changes without ulceration, and they have manifested no symptoms.

As the proctitis progresses in severity so do the symptoms. Tenesmus and urgency to stool are next most common. With ulceration the patient usually complains of pain and pressure in the rectum or pelvis. At this stage of the process constipation and even obstipation may be noted because stool passing over the involved area will cause considerable distress.

Symptoms of bowel obstruction manifest themselves as strictures develop, varying from crampy abdominal pains preceding defecation to obstipation and complete obstruction. With the Grade IV lesions with rectovaginal fistula the symptoms are obvious. Malnutrition is usually marked and pain severe in the inflammatory stages of the lesion.

Materials and Methods. The patients included in this study have been treated by the method described in detail by McKelvey, Stenstrom and Gillam.[1] Essentially this method consists of external irradiation delivered to the pelvis through five large fields which girdle the pelvis, namely, an anterior, two lateral and two posterior oblique portals. A single field is treated daily and the series of external irradiation is given over a period of about four weeks. This is followed immediately by a single radium application.

The external irradiation has been given to these patients with (1) a 400 KVP G. E. Maximar x-ray machine which produces radiation with a half-value-layer of 3.9 mm. of copper or (2) a Radioactive Cobalt Beam Therapy Unit which emits a beam of gamma radiation having a half-value-layer of 14.9 mm. of copper. Dosage from external irradiation to a small area on the anterior rectal wall has been in the range of 3,300 to 4,000 tissue roentgens in a period of approximately four weeks.

The single radium application at the end of the external irradiation series is usually carried out with 50 mg. of radium, using two containers of 10 mg. each in the intrauterine tandem, 10 mg. in each lateral arm of a Kaplan colpostat and 10 mg. in a central cork in apposition to the cervix uteri over a period of 100 hours, giving a total of 5,000 milligram hours. Calculated dosage from the radium indicates a very broad range varying with the distance from the radium to the anterior rectal wall. This range is from 2,500 to 3,500 roentgens in 100 hours.

Therefore, total combined dosage by simple addition and without considering tissue recovery in time is from 5,800 to 7,500 roentgens to tissue on the anterior rectal wall in a period of approximately five weeks.

Pathogenesis. The intestinal mucosa is unusually susceptible to damage from irradiation.[10] It has been estimated by some[7] that the mucous membrane of the rectum will tolerate only two-thirds the amount of irradiation tolerated by the normal uterus. Patients also manifest variance in their individual susceptibilities to the same dosage of radiation.

The primary damage in factitial proctitis appears to be to the smaller branches of the hemorrhoidal vessels with thrombotic occlusion and subsequent infarction with mucosal ulceration of the involved segment. Spasm further increases the ischemia and delays the attempt at repair.

Ferguson[7] demonstrated in acute factitial proctitis that there was a mucous degeneration of the rectal glands with round cell infiltration and hyperemia and edema of the tissue of the rectal wall. Sections of strictures showed marked atrophy of the mucosa with a slight degree of fibrosis in the submucosa and muscularis. The chief thickness of the bowel wall and probably the stenosis is the marked degeneration with swelling and edema of the muscular coat, especially the circular layer.

TREATMENT

An attempt is made to determine whether or not some therapeutic agent might contribute to the healing of this chronic and disabling process. Buie[3] has stated that few patients with factitial proctitis will heal spontaneously in less than fourteen months and generally not before two years have elapsed. Healing then only occurs if the primary disease is arrested.

During the past three years the author has been impressed with the oral administration of Compound E in the treatment of some cases of ulcerative colitis, particularly when early stricture formation exists. Although no explanation has been proposed as to the mechanics of healing, it was thought advisable to take the cue and administer Compound E orally and topically to twelve patients with irradiation proctitis. The results were inconsistent and difficult to interpret. Several patients with deep chronic rectal ulcerations healed in eight- to ten-week periods but in the main, results were disappointing.

TABLE I

Classification	No. Patients	Healed	Delayed Healing	No Change	Perforation
Grade I.....	16	14	2	0	0
Grade II.....	11	7	2	1	1
Grade III.....	8	4	1	3	0
Grade IV.....	5	1	4	0	0
	40	26	9	4	1

During the past year Compound F has been used topically in forty patients with irradiation proctitis and the results in most of the patients have been very gratifying. The majority of the Grade I and II lesions have largely disappeared in approximately two months after a regimen of topical administration of hydrocortisone to the lesion. In the Grade III and IV lesions the sloughs and exudates have appeared to "clean up" and several early strictures have apparently relented to the extent that a proctoscope could be passed through them.

Method. Since the majority of the lesions of factitial proctitis involve the upper rectum (10 cm. average location), the topical application of a medication by the patient to this level is difficult to achieve. Suppositories containing 50 mg. of hydrocortisone each were tried. Saline instillations of similar dosages were also tried. The most effective method of administering hydrocortisone to this level of the rectum, in our hands, has been by the use of a water soluble jelly base and a 4 inch rectal applicator. The patient is instructed to cleanse the rectum twice daily with a warm saline solution introduced by means of an infant bulb syringe, then to apply either 50 or 75 mg. of hydrocortisone jelly with the long rectal applicator, according to the level of the lesion. Particular effort is made to describe to the patient the sacral curve of the rectum and the effective level at which to apply the jelly.

Results. Forty patients with varying degrees of factitial proctitis have been treated and followed proctoscopically during the past year. The patients were seen at two- to four-week intervals and the results recorded by endoscopic photography. The results are shown in Table I.

The term "healed" implies that the lesion has largely disappeared during an eight-week period of treatment. The Grade I lesions, the proctitis without ulceration and stricture formation, responded almost entirely to the topical applications of hydrocortisone. Several persisted for three or four months, but eventually they all presented a fairly normal-appearing bowel except for some slight residual scarring.

"Delayed healing" implies that a three- or four-month period of treatment was required before healing occurred. The Grade II lesions, proctitis with ulceration, were somewhat more resistant to treatment as Table I indicates. Nevertheless, seven patients presented healed ulcerations in less than two months and two patients healed their rectal ulcerations in less than four months. One patient with carcinomatosis has failed to heal her ulceration after six months of therapy, and perforation of a factitial ulcer with a subsequent rectovaginal fistula developed in a patient on topical hydrocortisone. It must be explained that this patient did have a fairly deep-seated ulceration and inadvertently was temporarily lost to follow-up. When she reappeared in the clinic two months later, during which time she faithfully applied hydrocortisone to the rectal ulceration, a rectovaginal fistula was apparent.

It can be noted in Table I that four patients with strictures (Grade III) are classified as "healed" and one patient responded with "delayed healing." These were all early strictures. Prior to treatment a small ½-inch sigmoidoscope could not be passed through the narrowed bowel and several patients complained of signs of partial large bowel obstruction. These symptoms subsided and a ¾-inch sigmoidoscope could be passed through the strictures of five patients reported as "healed" after hydrocortisone therapy. Actually some degree of scar tissue persisted in all of these patients, but strangely enough the early strictures seemed to undergo dissolution. The

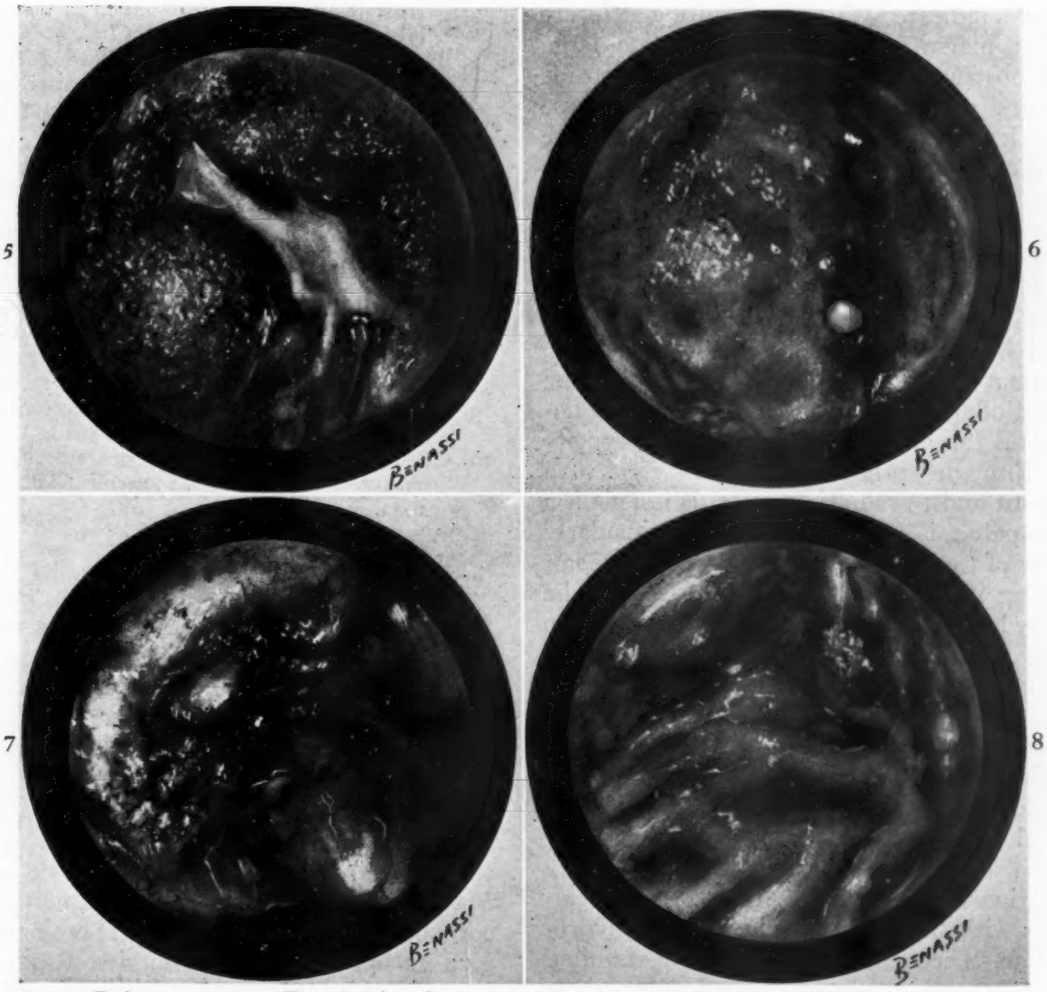


FIG. 5. Before treatment. FIG. 6. After four weeks of topical hydrocortisone. (Patient is fifty-seven year old white woman with Grade II factitial proctitis which developed eleven months after x-ray and radium therapy for a League of Nations Stage II squamous carcinoma of the uterine cervix.) FIG. 7. Before treatment. FIG. 8. After eight weeks of topical hydrocortisone. (Patient is a forty-nine year old white woman with Grade II factitial proctitis which developed six months after cobalt beam and radium therapy for a League of Nations Stage II squamous carcinoma of the uterine cervix.)

chronic strictures of long standing and particularly those in the colostomized patients were little affected by treatment. (Figs. 5 to 8.)

COMMENTS

Systemic Absorption. The three patients who were followed with eosinophil counts and 17-ketosteroid urinary excretion studies did not indicate that there is an appreciable absorption into the blood stream by the administration of 75 mg. of hydrocortisone rectally twice daily.

Bowel Perforation. This possibility must always be borne in mind. Topical hydrocortisone therapy to deep-seated bowel ulcerations involving the muscularis should be avoided. All patients receiving this regimen of therapy must be followed with frequently repeated proctoscopic examinations.

Control Series. Several authorities point out that factitial proctitis is a self-limited process as far as the Grade I and II lesions are concerned, yet usually fourteen- to twenty-four-month periods elapse before spontaneous healing occurs. This represents a preliminary report and a control series will be reported in detail in the final version. To date ten patients with Grade II lesions have shown little inclination to heal their ulcerations in nine-month follow-ups.

Surgery. It is not the purpose of this report to propose medical therapy where surgery is obviously indicated. A diversionary colostomy

is always indicated when the resulting stricture is of a magnitude to cause complete large bowel obstruction. However, if the stricture is early and the obstruction incomplete a course of medical therapy should be tried. It is the author's belief that several patients in this group have been saved colostomies by such a conservative regimen.

Much speculation exists as to the optimum time for definitive surgery on Grade III and IV deformities, and some even doubt that surgery should be attempted in view of the chronic inflammatory aspect of these lesions plus the element of tissue damage secondary to irradiation. It is this author's belief that some type of "pull-through" procedure can best cope with Grade III and IV radiation proctitis lesions if a frozen pelvis is not encountered, particularly in view of the contraindications to local repair following irradiation. These lesions should be "cleaned up" as much as possible prior to surgery.

SUMMARY

The various degrees of factitial proctitis are classified into four groups with particular reference to their adaptability to medical or surgical treatment.

This preliminary survery indicates that early irradiation lesions may improve in many instances with the topical applications of hydrocortisone. Those patients with long-standing changes involving the rectum are less favorable candidates for medical treatment.

The possibility of encouraging bowel perforation with steroid therapy must be borne in mind. This is presented in hope that other workers will be stimulated to investigate this difficult problem.

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DISCUSSION

Robert S. Mechling (Pittsburgh, Pa.): Dr. Sherman is to be commended for the work he has done on this most distressing condition. There are only two other reports in which as large a number of cases has been studied. No article was found in which the patients had been so systematically observed and treated.

This work appears to be original in its field. There is no reference in the literature to a similar study. At the University of Pittsburgh Medical Center we have not as yet used hydrocortisone for factitial proctitis. Therefore, I can give no opinion as to its merits. Research in other fields has shown that cortisone will cause dissolution of granulation tissue, soften newly deposited fibrous tissue and inhibit the formation of dense scar tissue in many instances. This would seem to be confirmed by the author's work.

The word of caution about bowel perforation in patients having hydrocortisone applied directly to ulcers should be stressed. In these cases the normal protective fibroblastic response is inhibited. On the other hand, since there is minimal systemic absorption of the drug when topically applied, the important sealing off of the neoplasm by fibrous tissue barriers resulting from irradiation should not be impaired.

Most authorities on this subject agree with Dr. Sherman that cases of early stricture with incomplete obstruction should be given a trial on medical therapy. Early colostomy will prevent dilation of the involved area by stool and so encourage the development of a more

complete obstruction. When surgical intervention was necessary at our Medical Center Hospitals for impending obstruction, either anterior resection of the sigmoid or rectosigmoid area or colostomy was performed. We have had no experience with abdominoperineal proctosigmoidectomies in these cases.

This paper should encourage all of us to work more closely with our x-ray and gynecologic colleagues. It should stimulate us to continue and expand this work started by Dr. Sherman to minimize postirradiation complications until a way is devised to prevent them.

Pilonidal Disease

REVIEW OF THE LITERATURE WITH COMMENTS ON THE ETIOLOGY, DIFFERENTIAL DIAGNOSIS AND TREATMENT OF THE DISEASE

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PILONIDAL disease is not a rare surgical condition and yet it has become to some extent a surgical problem in that the recurrence rates as reported in the literature are frequently embarrassingly high. Furthermore, it is quite evident that the origin, development and diagnosis of the disease is not clearly understood. This thesis was undertaken in an attempt to clarify these points.

Pilonidal sinus is a common skin defect usually located in the midline of the body over the sacrococcygeal region. It is congenital in origin and becomes symptomatic only when infected. When infection supervenes, pilonidal disease presents all the signs and symptoms of acute abscess. The telltale ostia of the sinuses are readily discovered in most cases and pus can frequently be expressed from them. If drainage through these openings is inadequate, the inflammatory process may burrow in any direction through the subcutaneous fat erupting elsewhere to form one or more secondary openings. The material which exudes from these infected sinuses consists of bloody pus, foul smelling sebum and occasionally hairs.

DIAGNOSIS

"Without a diagnosis there can be no intelligent treatment," and Mathews' other didactic admonition "look and see" constitute the alpha and omega of the differential diagnosis of pilonidal disease. The following excellent list of conditions which produce sinuses was compiled by N. D. Smith[144] and should be known and understood in order to prevent mistakes: (1) Spontaneous infectious processes: fistula-in-ano; incomplete fistula-in-ano (ischioanal abscess); fistula with anal fissure; rectovaginal fistula; infected lymphatic vessels; periurethral abscess. (2) Lesions arising in glandular structures or cysts: pilonidal disease; chromaffin bodies; multilocular cysts; bartholin cysts; pyoderma; dermoids; hidradenitis suppurativa. (3) Etiology (unusual): tuberculosis; actinomycosis; osteomyelitis; penetrating injuries; lymphogranuloma inguinale; carcinoma, epithelioma and lymphosarcoma. (4) Pseudosinuses: congenital anal dimple; postanal dimple; comedones; enlarged hair follicles.

In order to complete this list the conditions of simple furuncle, sebaceous cyst, epidermoid and the various grades of meningocele and other low spinal cord defects must be added. Most commonly, confusion in diagnosis is seen in the consideration of the condition of coccygeal dimple. The latter was described and studied by Dunlop[40] who remarked that a deformed coccyx usually was palpated beneath this anomaly. In most instances the dimple is located 1 or 2 mm. cephalad to the tip of the coccyx and the opening is comparatively broad and shallow. When probed, the direction of the dimple is caudad.

Reference to the entity of hidradenitis suppurativa as noted in Smith's list cannot be dismissed without further discussion. It is my opinion along with others that hidradenitis is not as uncommon an entity as is generally believed. The many and repeated references to the "hairy, obese, glandular type" of persons are just as compatible, if not more so, with hidradenitis subjects as with individuals suffering from pilonidal disease. It is my opinion, along with others, that pilonidal disease and hidradenitis suppurativa are more frequently confused than any other conditions that produce sinuses in this area. Examination under anesthesia and even histologic examinations of excised tissues may be necessary to clarify the ultimate diagnosis.

COMPLICATIONS

Pilonidal disease is chronic, recurrent and frequently progressive in that the area of involvement in inflammatory reaction may spread with each recurrence. A particularly

virulent infection may produce a dangerous cellulitis. Actual osteomyelitis of the sacrum is a very rare complication since the heavy fascia overlying the vertebrae is apparently very resistant to infection. No records of septicemia or death following infection have been found in the literature. Following operation, delayed healing is common and persistent drainage is frequently seen. Meningitis as a complication has been described by Moise[109] and by Ripley and Thompson.[131] In both of these papers the cases reported occurred in children, and the squamous epithelium-lined tracts were located in the high sacral or low lumbar region. Hurwitt[78] described subphrenic abscess following surgical care. Goldman and Kalow[62] have reported a case of pilonidal disease complicated by a basal cell epithelioma. This is the only report in the literature of a cancer occurring in conjunction with pilonidal disease. Metaplasia is a common finding on microscopic examination of these tissues but real neoplastic change has not been seen in specimens examined by us.

HISTORY

Most authors credit A. W. Anderson's[1] article published in 1847 as being the first description of this disease to be found in the literature. Fourteen years before this time, however, Herbert Mayo[106] described a case in which a young woman presented herself for treatment of a sinus which opened an inch behind the anus and ran upward over the sacrum a distance of 5 inches. The sinus was superficially located, did not connect with the rectum and must be regarded as an instance of pilonidal disease.

Curiously enough Dr. Mayo successfully treated this case by opening the tract and letting it granulate to the surface. J. M. Warren in 1854 was the next author to describe the condition and in 1877 he published a report[166] of another case and presented a theory regarding its etiology. Since that date many thousands of cases have been reviewed and hundreds of authors have contributed to the knowledge of this subject.

ETIOLOGY

Warren[166] in 1877 described the condition of pilonidal disease and opined that it was caused by a hair which grew inward and coiled as it grew. This produced a cavity lined with hair. A second theory was evolved by Hodges[71] in 1880 who believed that in a delayed or faulty closure of the neural canal and skin some epithelial bits were caught below the surface and remained to develop later as sequestration cysts. With a few alterations this theory caught the professional fancy and since has been backed by most authorities on this subject. A third theory may have been foreshadowed by Tait in 1877, who stated that the disease was due to a hereditary cicatrix of the spina bifida by which the human tail was lost. Tourneaux and Herrmann in 1887 studied serial sections of the human embryo and described the coccygeal medullary vestigis of the neural canal located in the subcutaneous tissues over the low sacrum and coccyx. These were hit upon as the precursers of pilonidal disease. Mallory[103] in 1892 confirmed their findings. Subscribers to this theory became numerous and powerful. Walker and Bucy[164] bring this theory to its logical conclusion by describing seven cases, including one of their own, in which a patent tube existed between the skin and spinal canal. These authors claim that the origin of this type of defect was due to an incomplete cleavage between the cutaneous epithelial ectoderm and the neuro-epithelial ectoderm in the first embryonic month. Thus the neural tube carried down with it a narrow invagination of skin, clothing it with a mesodermal covering continuous with its own connective tissue, the meninges. Stone[148] in 1924 quoted a personal communication from Streeter of the Carnegie Institute in which this noted authority on embryologic development stated that the coccygeal medullary vestige has no opening communicating with the skin. The vestige consists of differentiated cells which could not give rise to skin. In view of these findings, Streeter believed, as did Stone, that pilonidal disease was due to a special local downgrowth of epithelium originating from true skin and not from the medullary groove. Streeter[152] had previously described the tubules of Herrmann and Tourneaux in an exposition on the filum terminale and cauda equina and had found these vestiges to be lined with columnar epithelium.

Lannelongue in 1882 proposed a fourth and ingenious theory which was quickly backed in the same year by Heurtaux. This idea presupposed a firm fibrous connection between skin and bone in the sacrococcygeal region during

early embryonic life. Later when mesodermal tissues grew in from the sides elevating the skin all round these attached spots, clefts and even cysts were formed lined with dermal elements. Wéndelstadt in 1885 agreed with this theory but added that the connection between skin and bone was probably a remnant of the spinal cord tip and thus produced a traction dermoid.

Stone[148] in 1924 suggested that a special local downgrowth of epithelium originating from true skin was the cause of pilonidal disease. This fifth theory repudiated the theory that the disease was due to a remnant of the medullary groove. Quoting Streeter, Stone argued that the coccygeal medullary vestiges had no opening communicating with the skin and that they also consisted of cells which had differentiated enough so that they could not revert to or develop into true skin. In 1931, Stone advanced the foregoing theory again and suggested that these downgrowths of skin might be similar to that observed in modified apocrine glands such as the breast. He believed that they could be related to the preen glands of birds and to the scent glands of some mammals. Bloom (1930)[17] was given as added support. Fox[52] in 1935 added to this by saying that the ectodermal downgrowth developed during the third and fourth fetal month and was probably a skin appendage developing to maturity in or at puberty. Patey and Scharff[118] have suggested that pilonidal sinuses are foreign body granulomas produced as a result of penetration of the skin by hair.

Most of these theories regarding the etiology of pilonidal disease have some basis on facts. It is highly probable that some investigators have inadvertently included other entities such as the pyodermas, spina bifida and coccygeal dimple in their series. The author is strongly inclined to believe that true pilonidal disease is an embryologic skin defect and as such can be classed with harelip as a simple instance of halted development.

With the foregoing remark it is seen that the author has aligned himself in this seventy-five year old argument with Hodges who first advanced such a theory of origin in 1880.

INCIDENCE

Most observers agree that skin defects over the sacrococcygeal region are not uncommon. Bane in 1917 remarked that the irregularities are commonly seen in children. Biegeleisen[13] in 1939 stated the one of every three infants and 4 per cent of the adults examined had defects in this region. Heurtaux in 1882 found skin defects in from 4 to 5 per cent of the 960 adults examined.

In 1938 Johnson and Levingston[80] found that 3.4 per cent of 1,000 mentally defective patients had abnormalities of the skin over the sacrococcygeal region. It has been noted that in children coccygeal dimples are much more common than pilonidal defects. Dulligan[38] writing in 1929 and Kooistra[88] in 1942 agree that a case of pilonidal disease is found in each 1,000 general hospital admissions. I find pilonidal disease or sinus once in every twenty-six proctologic consultations.

Age. The age at which this disease makes itself evident to the patient by becoming infected coincides generally with the age of puberty.

Race. Mostly all of the patients with pilonidal disease are Caucasian. However, when this disease is found in members of other races, it must not be considered unusual. I see less Negroes with pilonidal disease than I do Caucasians but this lowered incidence is directly proportioned to the total number of each race that I have observed.

Hodges writing in 1880 pointed out that his patients were usually excessively hairy. This description has been stereotyped through the literature and many references have been made to the "hairy, obese, glandular type" of person. That pilonidal disease has a familial tendency has been noted in the literature by Hayter,[63] Anderson,[2] Kallet, Dunlop[40] and Goldberg and Bloomenthal.[61] Since congenital defects are so commonly multiple it has been suggested that other deformities would be in conjunction with pilonidal disease. This theory has not been substantiated.

Sex. Pilonidal disease is preponderantly an affliction of the male. Hodges in 1880 recognized this and his early observation has been confirmed many times since.

Location. Lannelongue in 1889 determined exactly the location of the disease in relation to the underlying bones and found that in 40 per cent of the patients examined the lesion was directly over the sacrococcygeal articulation; 30 per cent were located somewhat caudad to this point and an equal per cent were slightly cephalad to the joint. Pilonidal disease is

rarely eccentrically located. I have seen sinuses lateral and even anterior to the anus.

Chronicity. The chronic recurring nature of pilonidal disease coupled with a regrettable lack of knowledge of the pathology and the poor surgical technic employed have bolstered the figures given by various authors. Anderson,[3] Cupp,[35] Dulligan,[38] Horsley,[76] Smiley[142] and Stone have quoted actual figures on the percentage of their patients who had previous surgery. The extremes listed by them are 25 per cent and 100 per cent.

TREATMENT

Mayo in 1833 described for the first time in the literature a surgical treatment for this disease. Anderson in 1847 opened the sinus completely and cauterized the lining with silver nitrate. This method of treatment is still being used with slight modifications by Crookall who "scalps" the tract and then applies lunar caustic. Buie[26] advocates the same procedure as demonstrated in his described "saucerization procedure" used over 120 years ago by Mayo.

In attempting to demonstrate more clearly the limits of the disease process many ingenious methods have been proposed. One of the most common agents used for this purpose is methylene blue. This is mixed with benzene, acetone, alcohol, water or hydrogen peroxide and injected into the sinuses, staining them a brilliant blue.

Dunphy[42] deplores the use of methylene blue since he believes it frequently does not reach all of the diseased tracts. In addition, if too much pressure is exerted when the dye is injected, some of the coloring material will frequently escape from the confines of the sinuses and stain nearby normal tissues. Dunphy also states that in general the abnormal tissues are easily recognized grossly and, therefore, there is no reason to use definitive agents such as these.

The actual treatment of pilonidal disease is generally accepted as being surgical. It must be managed as is any other acute or chronic inflammation. In the acute stage, drainage is indicated and curative procedures must be delayed until the tissues have demonstrated their ability to heal in the face of chronic infection. Curative treatment has been described using many different technics.

Biegeleisen and Block and Greene[16] have advocated sclerotherapy. Swinton and Cutler and Zollinger[36] laid the sinuses open before they employed the curet and sclerosing solution. Hodley[73] did the same thing using 95 per cent phenol as his escharotic agent. Landsman[92] simply opens and curets the tract. An ingenious procedure has been recommended by Moore who cores out the tracts by running a diathermy loop over a probe inserted into the sinus. After this procedure is completed, he closes the resulting wounds by primary suture. Maillard[101] has reported a cure after a course of treatment consisting of probing the sinus with an electrode activated by galvanic current. He uses 20 ma. for twenty minutes every five days for three months to effect this cure.

The most popular approach to the problem appears to be radical bloc excision of the diseased tissues. This is accomplished by wide elliptic incisions through the normal skin and subcutaneous tissues surrounding the lesion. These incisions are generally extended down to the heavy fascia overlying the sacrum and the whole block is removed. Rogers and Hall[134] believe that this sacrifices too much normal tissue and thus needlessly prolongs the healing time. They use a cautery and split the tracts with a linear incision and then remove the diseased halves thus revealed.

Buie probes the sinus and its ramifications and then, after incising down to the tracts, excises all overhanging wound edges to produce a saucer-shaped wound. The base of the wound is the floor of the tract which is not disturbed. He has aptly named this a saucerization operation.

It is the author's practice to excise the diseased areas sharply and scrupulously. The removal of areas of neighboring normal tissue is not advocated. However, the final wounds are purposely designed to have gently sloping edges, avoiding undercutting. Wound edges at or about 90 degrees to the surrounding surface skin are not desirable. Many surgeons leave these large wounds open and allow them to heal by granulation. Others use complete primary closure. Due to the large wounds that often result after bloc excision, several ingenious single or double pedicle flap procedures have been proposed to enable successful primary closure. Several intriguing undercutting operations have also been described to effect this end.

Scrupulous and painstaking postoperative

care is extremely important if good results are to be expected; either open or closed surgical technic may be used. Rosser and Kerr, and Blaisdell detail the care of open wounds at length. Personally, it has been found that a clean, dry wound washed several times a day with soap and water heals fastest. Overly exuberant granulations can easily and painlessly be excised if necessary. Postoperative care of the closed wound varies with the technics used but, again, must be meticulous.

Kleckner has probably presented the most ambitious evaluation of methods and results in the treatment of pilonidal disease. His composite reports on over 4,000 patients reveal conclusively that the open operation is the procedure of choice. The recurrence rate is lower, length of hospitalization is less and the patient is back to normal habits in a shorter time.

In the last few years, I have tended to minimize the use of suture material using it only, and very sparingly, for hemostasis after all other measures have been tried and found inadequate. Blaisdell's article, published in 1947, aptly states that "recurrence in the open wound from any cause is permanent delayed healing . . . recurrences following the open method tend thus to obtain accurately in one's own statistics; those following the closed method too frequently come to light in someone else's figures."

CONCLUSIONS AND COMMENTS

1. The author is convinced that pilonidal disease originates as a result of imperfect fusion of the ectoderm in the mid-sacrococcygeal region during the early weeks of embryonic life.

2. The disease becomes active and symptomatic through the agency of trauma or the irritation of accumulating epithelial debris and hair coincidentally with the generalized increased activity of the skin at puberty.

3. It is important to differentiate pilonidal disease from coccygeal dimple, pyoderma and meningeal defects. The differentiation is usually easy if one keeps in mind the varying clinical, gross and microscopic characteristics of each entity.

4. The author has studied the microscopic characteristics of 133 specimens of pilonidal disease excised in the course of consecutive operations performed over a three-year period. Of these specimens 40 per cent contained no epithelium or hair, and all of the patients had been operated upon at least once previously. It must be inferred, therefore, that the persistence of the disease was due to poor operative technic or improper postoperative care.

5. The author has conducted a statistical review and follow-up research of 499 cases and found the following interesting percentages regarding persistence of the disease:

Type of Operation	Cases	Recurrences	Per cent
Bloc excision—closure with drain	93	22	24
Bloc excision—complete closure	43	8	19
Bloc excision—partial closure	38	7	18
Exteriorization (saucerization) procedure	34	2	6
Bloc excision—non-closure	289	12	4

Surgical excision is the treatment of choice. It would seem from the foregoing that it is unwise to close the defect resulting from the operation. This is undoubtedly due to the regionally poor blood supply, infected character of the wound, inability to obliterate dead spaces completely and the almost universal necessity of using tension on the tissues and suture material in attempting to coadopt the edges of the wound. It is my firm belief that the advent of myriads of antibiotics and tissue digestants has not placed surgery for pilonidal disease beyond the pale. Retribution in the form of persistence of disease is certain when the hard-learned precepts for prompt and permanent healing by primary and secondary intention are ignored or by-passed. It is of incidental interest that the advocates of excision and closure procedures prefer and for the most part limit these procedures to selected cases in which there is no evidence of infection at the time of surgery. The author has long believed that many such cases may be ignored since the disease could indeed be completely "burned out" and need no treatment at all. I believe that the risk of neglect in such cases is small.

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Hidradenoma of the Perianum

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Adenomas of apocrine sweat glands, hidradenomas, are primarily important because of marked similarity to adenocarcinoma which may cause the patient to be subjected to needless extensive surgery.[2] Cooper and McDonald[7] described the first case found at the anus; Ackerman[1] reported a case in the anal canal which was first misdiagnosed as carcinoma of the rectum invading the anus. The case now presented, so far as could be determined, is the third perianal hidradenoma to appear in the literature. Most of these tumors occur in the labia majora and less frequently in the labia minora. They apparently can arise anywhere apocrine sweat glands are found. True adenocarcinomas of sweat glands, both apocrine and eccrine or ordinary sweat glands, do occur and their distinction will be discussed.

HISTORICAL SKETCH

The first description of hidradenoma was presented just before the turn of the century, but Pick[19] in 1904 separated his case from other sweat gland tumors and gave them the name of hidradenoma. Other cases then followed in the foreign literature, but it was not until 1915 that the first case was described in this country by Outerbridge. Later reports followed in the literature until eighty had been reported[9] by 1947, and the total was raised to about one hundred[3] by 1950.

There is agreement on the gross and microscopic observations in hidradenoma, but there is disagreement concerning the malignant potentialities of the condition.

SIGNS AND SYMPTOMS

A hidradenoma, which is usually asymptomatic, may be solid or cystic, resembling a small sebaceous cyst or a fibroma and varying in size from 0.5 to 1.5 cm. in diameter. Two hidradenomas in one patient have been reported. The tumors, according to Lennox, Pearce, and Symmers,[14] usually contain some mucin, though in markedly varying amounts which may be barely detectable grossly. Mucin is not present in normal sweat glands in man but is found in similar glands of many lower animals.[14] It is not amazing that the type of secretion should change in abnormal function when we consider the versatility of secretory cells of apocrine glands. Within one species we find them specializing into apocrine sweat glands, ceruminous glands, eyelid glands of Moll and parenchymatous breast tissue.

These tumors are freely moveable under the skin but are attached to it. Apocrine sweat glands are situated deep in the dermis. At the site of attachment the skin may be slightly raised and discolored, or a red center may be evulsed through a small ulcerated opening in the skin, in which case there may be a slight bloody or clear discharge. In at least one case with an evulsed red center, the inguinal nodes were moderately enlarged and the surgeon did a wide excision of the tumor and an inguinal gland dissection. The diagnosis was benign hidradenoma and, of course, no metastasis.

Apocrine sweat glands form with the hair follicles which produce the hairs that are a secondary sex characteristic, and this fact accounts for the presence of the glands primarily on the pubis, vulva, perianum, axillae and around the areola of the breasts. Although these glands are formed with the hair follicles, the ducts may wander from the follicle and open directly on the skin. Their proximity to hair follicles explains their partial inclusion of, or lying adjacent to, sebaceous glands in some cases. These are the odoriferous glands, supposedly concerned with sex stimulation in many lower animals, and in them are much more numerous. The glands are about twice as numerous in women as in men, and hidradenomas have been reported much more frequently in the female. Negroes have about three times as many apocrine sweat glands as Caucasians, but no case of hidradenoma has ever been reported in a Negro; however, three cases of sweat gland carcinoma have been reported in colored patients.[20]

Apocrine sweat glands do not begin to func-

tion until puberty but continue to function in old age. The youngest patient reported[18] with hidradenoma was a female age twenty-eight, the oldest was eighty. In women, function of the glands is reduced during the intermenstrual period but increased in the premenstrual period and during pregnancy. The rate of function apparently does not effect the tendency to formation of hidradenoma since most of these tumors occur after the age of forty. Since hidradenitis suppurativa is an infection of eccrine or ordinary sweat glands, this condition has no bearing on hidradenoma.

TYPES OF SKIN GLANDS

The apocrine glands differ from eccrine or ordinary sweat glands in origin, secretory function and characteristics of the secretions. They form with sebaceous glands and hair follicles from the primary germ of epithelium and are therefore formed from already differentiated epithelium, while eccrine glands are formed from undifferentiated epithelium.

Skin glands are of three types: merocrine, apocrine and holocrine.[18] Merocrine glands are characterized by eccrine sweat glands in which epithelial gland cells remain intact during their secretory function. Apocrine glands include in their secretion some of the cell cytoplasm, thereby partially destroying themselves in the process. In holocrine glands the epithelial gland cells degenerate completely and make up the gland secretion as is the case in sebaceous glands.

HISTOLOGY AND HISTOPATHOLOGY

Apocrine glands are small acinar bodies of epithelial origin and the lining epithelium is multilayered. Lining the gland is a layer of culumnar epithelial cells and there is an outer myoepithelial layer.

The hidradenoma consists of a cluster of irregularly grouped acini, lined by columnar epithelium composed chiefly of large cells with clear cytoplasm. In many places there are clearly multiple rows of epithelium and in addition there is often a thin myoepithelial layer which lies next to the membrana propria.

The combination of multilayered epithelium and an adjacent myoepithelial layer, imparting the appearance of pseudostratification in a tumor of adenomatous character, readily serves to simulate carcinoma. The histologic confusion is further heightened by the propensity of hidradenoma to be papilliferous, the papillae forming an arborescent pattern.[12] The diagnosis of benignity rests entirely upon study of the intracellular characteristics.

Cellular morphology of malignancy evidently was present in cases reported by Eichenberg,[10] Novak,[18] Danforth[9] and Brattstrom,[4] but it is questionable that these malignancies developed from hidradenomas. Novak[18] said that in "only one case in the world's literature has malignancy appeared to develop in a hidradenoma. Even this assumption may not be correct as the tumor may have been definitely malignant from the onset. In other words, adenocarcinoma may arise directly from sweat glands just as the benign hidradenoma does, and not necessarily with the intermediation of the latter."

McDonald[17] reviewed thirty cases at the Mayo Clinic and considered twenty-eight to be adenocarcinoma grade I (Broder's classification) and two to be adenocarcinoma grade II. Quoting Anderson,[3] Gates, Warren, Warvi, Gray and and Novak, in three separate articles, accepted only two of McDonald's cases as malignant and declared the others benign and should be considered cases of hidradenoma papilliferum. From a clinical standpoint, all cases that have been excised locally have been cured since there have been no instances of recurrence and no metastases.

HIDRADENOMA AND INTRADUCTAL PAPILLOMA OF THE BREAST

It is interesting to note the similarity between hidradenoma and intraductal papilloma of the breast since apocrine glands are modified in the mammary region to form the parenchymatous portion of the breast. In their clinical behavior both of these conditions (1) are relatively benign and slow growing, (2) on very rare occasions may undergo malignant change, (3) usually are present for a long period before producing symptoms, (4) may produce a slight discharge which may or may not be bloody and finally (5) generally are cured on local excision. Histologically, one cannot ordinarily distinguish between the two conditions. One important difference is that intraductal papilloma of the breast is almost always multiple which rules out the possibility of their representing developmentally misplaced apocrine sweat glands.

CARCINOMA OF SWEAT GLANDS

Stout and Cooley[20] present thirteen cases of metastasizing sweat gland carcinomas, seven from the literature and six which they reported, and will be discussed here as a group representing these carcinomas. These cases were almost equally divided between the sexes. The youngest patient was age thirty years and the oldest eighty-four. Nine cases were in areas in which apocrine sweat glands are found; two in the skin of the axilla, three on the arm side of the axilla, two in the vulva, one in the scrotum and one in the region of the male breast. Of the other four, two were in the scalp and one each on the wrist and eyelid. All of the tumors described were red or purple in color. They involved the skin, and the majority formed single or multiple nodules which projected above the surface. In one case the tumor, which began as a nodule, was characterized by a thickened depression in the skin. Since dermatofibrosarcoma protuberans, keloid, metastatic carcinoma and even lymphosarcoma of the skin can all form red or violet nodules, all of these lesions have to be considered in the differential diagnosis. There does not seem to be any certain method of differentiation except by biopsy.

CASE REPORT

S. L. M., a fifty year old, white, married female, presented herself for examination because of the fear of cancer expressed by husband and friends whom she had told about the small growth near the anus. She first noticed the small growth about eleven months before and believed that at times it was slightly larger than at others. The little knot was occasionally mildly tender but there was no bleeding or discharge. Otherwise the history was not abnormal except that she had been nervous for several years and was chronically constipated, took daily enemas and had taken mineral oil daily for one year. Past history revealed she had a series of injections for hemorrhoids in 1943, a hysterectomy in 1946 and had a bursa operation on her left shoulder in May of 1953.

Examination revealed abnormalities relative to this presentation to be limited to the perianal region. Besides small external hemorrhoidal skin tags, there was noted a small nodule about 4 cm. from the posteroleft aspect of the anal verge. The nodule was about ¾ cm. in diameter, firm, round, apparently encapsulated, easily moveable underneath, but attached to the skin. The overlying skin was slightly elevated to a point with a questionable opening in the summit, and there was a very slight brownish discoloration.

After infiltration around the mass with procaine, the overlying skin and tumor were excised by sharp dissection and the wound was loosely sutured. Grossly the tumor had the appearance of a small benign, encapsulated fibroma.

Report from the pathologist, D. E. Fletcher, gave the diagnosis: Adenocarcinoma of the perianal region arising from a hidradenoma cysticum papilliferum, low-grade malignancy, and stated doubt that the tumor had been completely removed. Dr. Fletcher stated that presence of malignant changes would be questioned by some pathologists. Slides were sent to the Armed Forces Institute of Pathology for registration and diagnosis.

The patient was admitted to the hospital and a wider and deeper excision of tissue was made at the site of the tumor. The fat and fascia were approximated with interrupted chromic o sutures, but the skin edges were not sutured. She was seen last on the twelfth postoperative day and the wound was well healed. The pathologist's report showed one small group of tumor cells partially enclosed in fibrous capsule.

Report from the Armed Forces Institute of Pathology diagnosed the tumor as a hidradenoma papilliferum and said, "Although the lesions are quite cellular and papillary, they have followed a benign course. No evidence of carcinoma is noted in the present lesion."

COMMENT

The rarity of hidradenoma of the perianal region may be more apparent than real. Women have learned to be very cancer-conscious about their genitalia and hasten to have the smallest lesion on the vulva examined, which may explain the large majority of hidradenomas being discovered on the labia. Small "growths, knots, or tags" at the anus are usually ignored by patients unless they cause symptoms. Review of autopsy reports would be of no value in establishing the incidence, because at routine autopsies, inspection or palpation of the perianum is usually not done.

Sweat gland carcinomas show no particular predilection for the vulva. Of the nine cases, reported by Stout and Cooley, that were present in the sites where apocrine sweat glands are most prevalent, only two were on the vulva.

There is no definite indication that benign hidradenomas are premalignant lesions, and the author believes, after reviewing the literature and the report from the Armed Forces Institute of Pathology, that this case presented is benign.

A small solid or cystic tumor, which is described microscopically as adenocarcinoma in a hidradenoma or adenocarcinoma of sweat gland origin, should be studied carefully before deciding to do extensive, definitive surgery for the cure of cancer. All unquestionable sweat gland carcinomas reviewed were nodular masses, red or purple in color.

SUMMARY

1. The subject of hidradenomas (adenomas of apocrine sweat glands) is reviewed.

2. Similarity between hidradenomas and adenocarcinoma of sweat glands is discussed.

3. Discussion is given to similarity of hidradenoma to intraductal papilloma of the breast and difference between hidradenoma and true sweat gland adenocarcinoma.

4. Apocrine sweat glands are described and distinguished from other skin glands.

5. A new case of hidradenoma papilliferum of the perianum is added to the literature.

6. Caution is suggested before performing definitive cancer surgery for cure of a so-called malignant hidradenoma.

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Presacral Cystic Tumors

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PRESACRAL tumors in general are uncommon but the proctologist has the opportunity of making the diagnosis more frequently than other physicians and therefore it is his particular responsibility to be aware of the more detailed diagnostic characteristics.

The incidence of presacral tumors in the general population is unknown, but Whittaker and Pemberton[1] reported that presacral tumors occurred at the rate of one in 40,000 admissions at the Mayo Clinic between the years 1922 and 1936. This compares favorably with the statistics reported by Ross[2] of one presacral tumor in 46,000 admissions at the Kings County and Meadowbrook Hospitals from 1939 to 1946.

In regard to distribution of the various types of tumors, Jackman, Clark and Smith[3] found in eighty-two cases thirty-two types which they considered congenital, which included fourteen chordomas, seven teratomas, five dermoids and six meningoceles. In eighteen cases the tumors were considered inflammatory and included abscesses, internal fistulas and oleomas. Twelve of the eighty-two cases were of neurogenic origin and five were osseous in origin. The remaining fifteen cases classified as miscellaneous included two lipomas, two myelomas, one fibrosarcoma, one leiomyosarcoma, four metastatic tumors and three indeterminate tumors. Mayo, Baker and Smith[4] on the basis of 161 presacral tumors have classified in order of frequency and occurrence five groups of tumors to be considered in the differential diagnosis. (Table 1.)

The general symptoms produced by presacral tumors vary greatly due to the associated pathologic changes. In many cases there may be no symptoms, the lesion being discovered by palpation alone. In other situations the complaints may be limited to indefinite low sacral pressure or backache, occasionally associated with constipation. The size and location of the lesion are more important in determining whether sciatica, rectal and urinary incontinence, or bony changes due to pressure or invasion will be present.

It is important to remember that teratomas and meningoceles are usually found in female infants or children; that dermoids and teratoid cysts occur at any age and are mainly limited to females; and that chordomas are usually found in adult males.

TABLE 1
DIFFERENTIAL DIAGNOSIS OF PRESACRAL TUMORS IN 161 CASES

Tumors	Cases	Per cent*
Congenital		
Chordoma	51	32
Dermoid cyst	29	18
Teratoma	19	12
Total	99	62
Inflammatory	18	11
Neurogenic		
Ependymoma	12	7
Neurofibroma	5	3
Neurilemmoma	1	1
Total	18	11
Osseous		
Giant cell tumor	5	3
Osteogenic sarcoma	4	2
Cartilaginous tumor	4	2
Osteoma	1	1
Total	14	8
Miscellaneous		
Metastatic carcinoma	4	2
Myeloma	3	2
Others	5	3
Total	12	7

* Based on 161 cases. Taken from Mayo, Baker and Smith.[4]

The roentgenographic findings of ependymomas, which are the most common tumors arising from the sacral canal, are sharp clear areas of bone destruction due to erosion by pressure. Chordomas and tumors arising from the body of the sacrum cause destruction by infiltration. Teratomas may produce anterior sacral wall erosion or may show evidence of bone or tooth formation within their masses.

This presentation is limited to observations in regard to seven presacral cysts seen in a ten-year period from 1943 to 1953. A review of the embryology of the region, the histology and treatment will be presented.

IMPORTANT COMMON FEATURES

All dermoid and teratoid cysts occurred in females within the age limits of eighteen months to forty-seven years. Hawkins and Jackman[5] give an incidence of three females to one male.

The most striking feature in each of the seven cases was the constant presence and location of a communicating sinus located in the midline posterior of the anal canal within the anal verge but always inferior to the dentate margin. Recent literature has failed to stress the specific location of sinuses in the anal canal that communicate with the main cyst. The presence of an opening in this location increases the chance for confusion with an anal fistula especially if the cyst has been drained by a previous incision posterior to the anus. This opening is characteristically funnel shaped in contradistinction to secondary fistulous openings.

Thin-walled cysts on the surface of the main cyst but not communicating with the main cyst were found in four of the seven patients. These thin-walled sacs contained milky or amber fluid and could easily be broken during the dissection; they result in recurrence. Histologic study of the walls of these cysts proved them to be composed of either columnar epithelium, columnar ciliated epithelium similar to the lining of the bronchial tree, or cuboidal epithelium almost identical with that found in anal ducts. In two cases there was evidence of extension by the process of rupture and infection extending as high as the sacral promontory. In two cases spontaneous rupture and drainage into the rectum occurred. In general, dissection was not difficult and was relatively bloodless except in the two cases in which there was an extension by rupture and infection. In the latter two cases parts of the levator ani muscle and sections of the rectal wall had to be excised. The prognosis is good if all tissue is removed.

The presence of a deformed coccyx was noted in all seven cases which consisted of a dorsal curvature of the terminal segment; this observation, in addition to the constant presence of a sinus leading into the posterior anal canal, led to a review of the embryology of the region.

EMBRYOLOGY

No one doubts that embryologic developmental defects are responsible for the presence of presacral dermoids, teratomatous cysts, pilonidal disease and chordomas. Figure 1 depicts diagrammatically the most important changes that take place during the third, fourth, sixth, seventh and eighth weeks of embryonic life.

The important changes during the third week show the neural groove (ectodermal anlage of the spinal cord) communicating with the cloaca by way of the neuroenteric canal at the level of the neuropore. The notochord is as yet unsegmented.

In the fourth week the notochord becomes segmented; the caudal portion of the neural tube has formed a rudimentary spinal cord; the neuropore has closed; the neuroenteric canal in the region of the coccygeal tip has disappeared leaving a section of the cloaca called the postanal gut caudad to the anal pit.

During the sixth week the cloaca is divided by the urogenital septum into the anlage of the rectum and bladder. The small circles and dots (Fig. 1) distributed between the caudal end of the neural tube and the postanal gut represent the degenerating cells of the neuroenteric canal. Schulte quoted by Bacon[6] has found evidence of these cells in forty fetuses examined.

In the seventh week the bladder and rectum are completely divided but the postanal gut is still apparent. The anal pit is prominent and the anlage of the genitalia are plainly visible.

The eighth week marks the fusion of the invaginating ectoderm of the anal pit and the hindgut to form the anal plate. The final diagrammatic sketch (Fig. 1) shows a persisting postanal gut sac to indicate how dermoid and tertoid cysts form their communication with the posterior anal canal. Actually no persisting sac was found in any of the embryologic sagittal sections that were reviewed. It was drawn in to emphasize how persistent postanal gut accounts for the presence of the communicating tract between presacral cyst and the anus. Further speculation could lead one to conclude that the secondary or daughter cysts found on the surface of dermoid cysts originate from cells remaining from an in-

completely degenerated neuroenteric canal. Pilonidal cysts with extensions anterior to the coccyx could originate in incompletely degenerated cells from the neuroenteric canal or postanal gut.

Two types of cysts were observed, namely, the multilocular (four cases) and the dermoid sac with or without daughter cysts (three cases). Figure 2 represents the dermoid sac type with thin-walled daughter cysts not communicating with the main cyst which is usually lined with squamous epithelium and contains material varying between thin, milk-like fluid to thick, putty-colored or chocolate-colored paste. The main cyst cavity communicates with the exterior in the posterior anal canal below the dentate margin and the external opening is characteristically funnel-shaped and lined with squamous epithelium. The thin-walled daughter cysts usually contain amber-colored liquid secreted by an epithelial lining of either columnar or cuboidal epithelium.

The dermoid type cyst may be quite large and may not be discovered until childbirth. One case in the series was seen after a craniotomy was done.* The multilocular type is a semi-solid cystic tumor composed of cyst walls lined with a combination of squamous epithelium, ciliated columnar epithelium almost identical with bronchial epithelium, cuboidal epithelium, columnar epithelium similar to colonic mucosa, and smooth muscle. In this

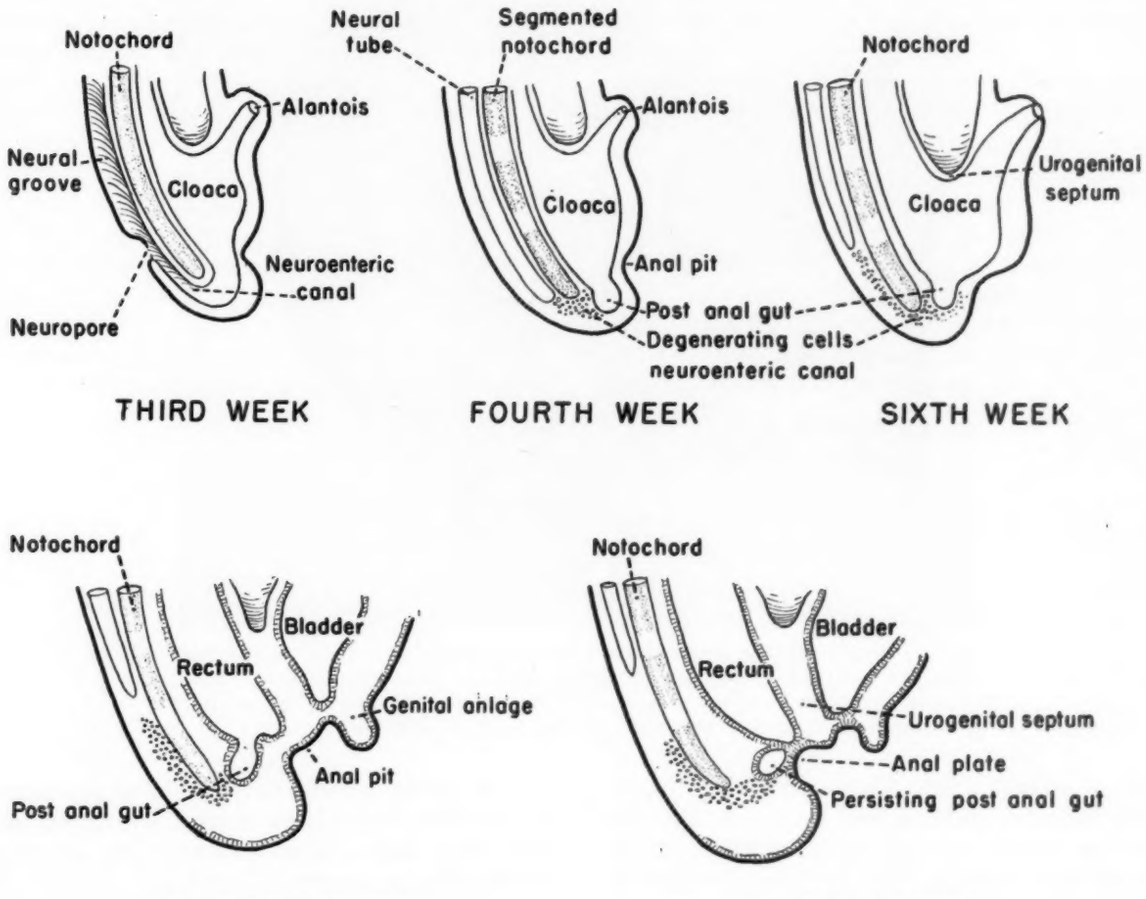

FIG. 1. Depicts diagrammatically the important changes that take place during the third, fourth, sixth, seventh and eighth weeks of embryonic life. In the final sketch labeled "eighth week" a persisting postanal gut sac is shown to indicate how dermoid or teratoid cysts form their communication with the anal canal.

* An added note of interest on this point was brought to my attention by Dr. Fred Bradford of Los Angeles in which he told of a presacral cyst obstructing the birth canal. The cyst was aspirated and the child successfully delivered by means of aspiration of the cyst by puncture of the needle through the rectum.

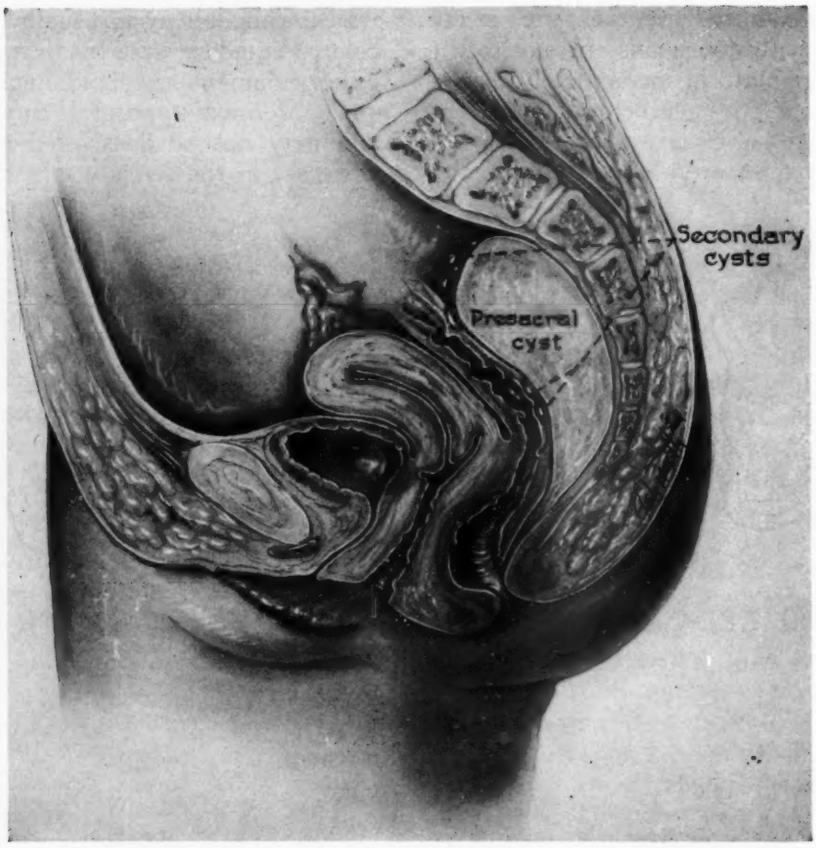

FIG. 2. Represents the dermoid sac type of presacral cyst with thin-walled daughter cysts which are not connected with the main cyst cavity. These daughter cysts are lined by cuboidal, columnar or ciliated columnar epithelium. The communication with the posterior anal canal is indicated.

type a probe cannot always be passed easily from the anal opening into a cyst cavity, but traction on the cyst will increase the dimpling at the anal opening. The multilocular type is more prone to rupture into the rectum or surrounding tissues.

CONCLUSIONS

1. The incidence and diagnostic characteristics of presacral tumors has been reviewed.

2. The gross and histologic characteristics of seven consecutive presacral dermoid and teratoid cysts are described which represent the number of such tumors seen in a private proctologic practice of 10,000 patients over a ten-year period.

3. An embryologic review attempts to explain the presence of a sinus communicating the main cyst cavity with the posterior anal canal, the presence of secondary cysts of enterogenous origin not communicating with the main cyst and a deformed coccyx.

4. It seems evident that presacral dermoids and teratoid cysts have their origin in an incompletely degenerated postanal gut or cells forming the neuroenteric canal.

5. Complete surgical excision must include the secondary cysts when present and the sinus communicating with the posterior anal canal.

6. Attention is called to the possibility that presacral cystic tumors may be confused with fistula.

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DISCUSSION

J. M. McIntyre (Indianapolis, Ind.): Dr. Laird has presented a good review of the recent literature, the symptomatology and embryology concerning presacral cystic tumors. It is interesting that Dr. Laird reports an incidence of one in fifteen hundred cases seen in a proctologic practice over a period of ten years; whereas other authors have reported an incidence of one in forty to forty-six thousand general hospital admissions. Probably we proctologists have a better opportunity to see these lesions.

Yeomans and Bacon in their respective texts report the presence of perineal and postanal sinuses connecting with dermoid cysts. However, no comment is made by either of these authors concerning the possibility of such a sinus being of congenital origin. Dr. Laird speculates that the communicating sinus between the presacral cyst and the anal canal is of congenital origin because the sinus in each of seven cases was lined with stratified squamous epithelium.

I think that dermoid cysts probably arise from the ectodermal portion of a persisting neurenteric canal, while teratoid cysts probably arise from a persisting postanal gut in addition to a persisting neurenteric canal.

It is important to think of the possibility of an anal sinus communicating with a presacral dermoid because fistulotomy will cure some simple anal fistulas whereas complete surgical removal of a presacral dermoid cyst and its communicating sinus is necessary.

Shock As It Pertains to Colonic Surgery

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IN a subject as broad as that assigned to me one cannot in any reasonable amount of space attempt to be comprehensive. Perforce, he must select certain points to discuss; and, even in those, to go into much detail would throw his presentation out of proper perspective. So I have selected a few aspects that I think are important, and I shall present my views on them in rather general terms.

I do not wish to become involved in the difficult and controversial matter of a definition of *shock;* yet in a discussion such as this, we must have some clear notion of what we are talking about, some common ground from which to proceed. So I shall take a somewhat arbitrary position, place the word "shock" in quotation marks, and use the term to refer to various decompensations of vital functions of the body insofar as they pertain to surgery of the lower bowel.

The first of these decompensations, or threatened decompensations, is that due to *acute blood loss.* This is a matter familiar to everyone, so we need spend little time on it. Blood losses during surgery range from insignificant amounts to relatively large quantities—as much as 1,500 or 2,000 cc. or more in prolonged, difficult operations. Unless we have taken the trouble to make actual measurements of blood in the suction bottle and on sponges and packs, most of us tend to underestimate the amount of blood lost during an operation.

Late signs of posthemorrhagic "shock" are well known (cold sweaty skin, low blood pressure, thready rapid pulse, panting respirations, etc.), but the alert surgeon and anesthesiologist will not wait for these dangerous signs of advanced and serious circulatory failure. The patient is able to compensate for slight losses of blood automatically; with larger losses the margin of compensation is narrowed. This stage of marginal decompensation, which is not particularly dangerous in itself because it can be corrected by prompt replacement of blood, should be recognized. It is characterized by slight gradual downward trend of blood pressure, a rising pulse rate, pronounced fall in venous pressure, decreased flow of blood through the skin capillaries and increased rate and depth of respiration, all of which signs are easily detected clinically. Measurement of the hematocrit is of little help here. Ordinarily we do fairly well in the recognition and treatment of this condition as long as the patient is on the operating table and is being followed minute by minute. The dangerous time comes when the patient starts to the ward again and is put back in his bed, the first few postoperative hours while he is observed only intermittently by the nursing staff and even more infrequently by the doctor. It is during this period that further undetected losses of blood may occur from the site of anastomosis into the bowel lumen, or into the tissues, or from the drained wound. It is at this time that the peripheral circulation is apt to be slowly but progressively depressed and the surgeon may return after some hours to find his patient in a dangerous state. It is fortunate for these patients if they can be kept during this critical period in a recovery room under the constant supervision of specially trained personnel.

The preventative treatment of posthemorrhagic depression of the circulation is simple and rational: Replace the lost blood, volume for volume, as it occurs. If this cannot be done or if it has not been done, at least give blood in the early stages of circulatory decompensation in amounts adequate to restore the circulatory physiology to normal. If through some mischance severe hemorrhagic shock has already

developed, give blood and give it fast—as fast as it can be run into a vein under pressure using a large needle. Use more than one vein if necessary. The risk of embarrassing a previously healthy heart by rapid intravenous transfusion is negligible. That organ is suffering from acute lack of blood—lack of blood to work on and lack of blood in the myocardium itself. Intra-arterial transfusions under these circumstances, some reports to the contrary notwithstanding, is unnecessary, is time-consuming, carries some risk and has no demonstrable advantages over intravenous transfusion given at the same rate. The giving of oxygen in posthemorrhagic "shock" is of secondary importance and cannot be relied on to help very much.

Another type of "shock" is due to acute losses of water and protein from the circulating blood. Extensive loss of red blood cells we can see; it forces itself on our attention. However, the loss of serum into tissues, into the peritoneal cavity, into traumatized tissues, or externally from drained wounds, is all too often overlooked. Yet potentially it is just as dangerous to lose plasma as whole blood. To these *localized* losses of serum must be added certain *systemic* acute losses of plasma protein, poorly understood but demonstrable, which result from severe trauma of many sorts including surgery.

When water and protein are lost from the circulation in small amounts, the body is able to compensate by shifting water from the extracellular compartment and by transfer of mobile protein slowly from protein "stores" to the blood stream; but when the losses are larger, such transfers cannot keep pace with demands and the peripheral circulation begins to show evidences of failure.

In this type of physiologic decompensation hypotension, although present, is less reliable as an index of severity of "shock" and one has to rely more on hemoconcentration. The early signs are: rising hematocrit, some fall in blood pressure, increasing pulse rate, increasing respiratory exchange and decreased blood flow in the skin. The face may be flushed rather than pallid, and the skin may remain dry and warm.

This sort of physiologic decompensation is dangerous because it so often goes unrecognized until late stages when the patient is suddenly discovered to be in serious condition or in fatal collapse.

Treatment is rational. These patients need plasma or plasma expanders such as albumin, dextran, or gelatin solution. It is a mistake to load these patients with salt solution or glucose solution. The water of crystalloid solutions runs out of the vascular bed as fast as it is put in, for there is insufficient protein to hold it in. If that sort of therapy is pushed, the tissues become water-logged. It is possible to produce pulmonary edema in that way. Paradoxically, these patients may need whole blood despite their hemoconcentration. To give a patient with a hematocrit of 60 a transfusion of citrated blood having a hematocrit of 38 will add needed plasma, reduce the hematocrit of his circulating blood and at the same time supplement a masked absolute shortage of red cells. A safe rule is to give the plasma or plasma substitute first; then, if the hematocrit of the peripheral blood falls below normal levels, administer whole blood also.

The amount of plasma or blood to be given under these circumstances is not easy to determine accurately. There are no formulas to go by. Bedside plasma volume determinations, serial measurements of plasma protein by the falling drop method and repeated hemoglobin measurements by photoelectric colorimetry are useful guides, but many hospital wards are not equipped to carry out those procedures. One must rely, rather, on physical signs and one's own clinical judgment, checked by an occasional measurement of the hematocrit. It is well to remember that in this plasma-poor type of circulatory failure, as in the pure oligemic type, prevention is better than cure and early treatment is more successful than late. Here also, administration of oxygen is apt to be of little benefit unless there happens to be coexisting pulmonary disease or respiratory embarrassment.

A third distinct sort of physiologic decompensation comes from gross disturbances of normal *fluid and electrolyte balances*. I am sure you must get tired of this ever-recurring subject. I do. Fortunately you, as colonic and rectal surgeons, are not bothered by it as much as your colleagues who deal primarily with the upper gastrointestinal tract. Nevertheless, fluid and electrolyte disturbances inevitably present themselves whenever there is acute diarrhea or vomiting, and often in cases of intestinal obstruction, gastrocolic fistula or extensive peritonitis. This is not an appropriate

occasion to enter into a discussion of this complex problem or into a consideration of the imperfectly understood mechanisms involved in any type of "shock." Suffice it to say, in both diagnosis and treatment of fluid and electrolyte imbalances, the recent trend is away from elaborate laboratory studies and application of convenient formulas. Many of the laboratory tests are being abandoned because they are too time-consuming and expensive, and fixed formulas have been discarded because in this disturbed physiology we deal with too many uncontrollable and indeterminate variables. The current attitude, rather, is to rely upon symptoms and signs, accurate records of fluid intake and output, measurements of urine volume and concentration, the simple bedside tests of blood chlorides and bicarbonate, and above all upon sound clinical judgment.

These three sorts of physiologic decompensation (those due to acute blood loss, to acute plasma loss and to severe fluid and electrolyte imbalance) are bad enough when they occur alone; but when they are superimposed upon long-standing depletion or debility of a patient, their danger becomes greatly enhanced.

I do not like the term "chronic shock," but it does indicate a state of depletion of one sort or another which is readily recognized. You see many patients of that sort in your field. These people are anemic and hypoproteinemic; their reserves of protein and glycogen are low; they maintain subnormal blood volumes; they have lost weight; their lives have become sedentary and their muscles soft and weak, including the myocardium. Poor appetite may have led to further depletion and sometimes to clinical or subclinical vitamin deficiencies. Spirometry shows reduced vital capacity and tidal exchange. Debility is particularly apt to occur, of course, in the old age group.

These patients present real problems to the surgeon. They are poor operative risks. Severe, acute physiologic decompensations are apt to develop when they are subjected to major operations, however carefully and skillfully performed. They are prone to infection and other postoperative complications.

If the primary disease permits, surgical treatment should be delayed until these patients can be gotten into shape for the ordeal. This cannot be accomplished in a hurry. Hypoproteinemia, for example, is a reflection of low protein reserves in the body generally which must be built up slowly. Chronically low blood volume cannot be corrected by one or two transfusions, just as underweight cannot be corrected by one or two big meals. It may take many days or many weeks to build these patients up, stock their metabolic stores, increase their resistance and immunity, and return their physiologic functions to a semblance of normal. However, the primary disease will not always permit such delays, or the disease itself may hamper all efforts at upbuilding. In the face of growing cancer of the rectum, gastrocolic fistula or active chronic ulcerative colitis, one may be forced to go ahead and risk whatever definitive surgery is indicated. However, when feasible to do so, it is the part of wisdom to spend all the time and effort required to get these patients into reasonably good shape before undertaking major surgery.

I would be remiss if I did not mention the frequent and sometimes dangerous disturbances of physiologic function due to the indiscriminate use of drugs. All of us can bear witness to the great improvement in anesthesia during the last decade. At the same time it has become popular among many anesthesiologists to give many drugs, one after another, to the same patient on the rather tenuous theory that one can obtain the cumulative good effects of all of them and somehow avoid the cumulative ill effects. Recently, I observed a patient who received scopolamine, morphine, atropine, sodium pentothal,® cyclopropane, oxygen, nitrous oxide, neo-synephrine,® curare, demerol® and quinidine—all within a few hours. Sometimes these drugs are given without consultation or even without the knowledge of the surgeon. I do not think that the current high incidence of operative and postoperative complications is unrelated to this machine-gun sort of medication. In my opinion, many patients are unnecessarily subjected to intratracheal intubation and "controlled" respiration. I believe that intravenous administration of glucose solution and salt solution and blood transfusions are often overdone during operation. My plea is for better understanding and closer teamwork between surgeon and anesthesiologist, and for greater caution in the employment of drugs and intravenous injections in surgery.

This caution should be extended also to overgenerous use of drugs on the wards, to the

excessive use of antibiotics, to the indiscriminate employment of ACTH and cortisone, and to the risky trial and error administration of proprietary drugs recommended by persuasive detail men.

If one were to take a group of healthy volunteers, put them to bed in a hospital and subject them to the full routine of surgical patients, omitting only the operation itself—let them undergo the strain of hospitalization, the interrupted sleep, the multiple examinations, a couple of transfusions, general anesthesia with intubation and all the attendant medications, postoperative intravenous therapy, gastrointestinal suction and numerous drugs of various sorts—members of this group, I contend, would leave the hospital a good deal less healthy than they were on admission. Some of them might really be ill; there would be some complications; and there might even be an occasional fatality.

What has all this to do with "Shock As It Pertains to Colonic Surgery"? A good deal, I think. "Shock" (as I use the term) is a serious disturbance of the vital physiologic functions for which the body is not able to compensate. The free and injudicious employment of many drugs before, during and after the operation, in at least some instances, and more often I believe than some realize, contribute to uncontrolled and dangerous disturbances of vital functions.

For the sake of completeness, we should also mention postoperative complications and their contribution to "shock."

One of the more common complications is atelectasis. When you operate upon a patient in Trendelenburg position and pack the intestines into the upper abdomen, the high-riding diaphragm hampers ventilation of the lung bases. This situation invites atelectasis. Although the abnormality usually does not manifest itself until twenty-four or more hours after operation, it frequently gets its start on the operating table. An alert surgeon, anesthesiologist or house officer will spot postoperative atelectasis in its early stages when it is relatively innocuous and easy to correct; neglected, its attendant pneumonitis spreads with the passage of every hour.

Another complication which may contribute largely to "shock" and is a danger inherent in your field is infection. Antibiotics and sulfa drugs have been a tremendous boon here, both in prevention and control of infection. Unfortunately the excessive, irrational use of antibiotics the country over is resulting in an increased incidence of infections from drug-resistant microorganisms. It is well in this connection to remind ourselves that before the advent of antibiotics master surgeons were able to do colonic surgery with low incidence of infection. Their secret lay in meticulous workmanship, gentle handling of tissues, good hemostasis, accurate anatomic repair and preservation of physiologic function. These fundamental principles of good surgery, sometimes lost sight of nowadays, are still as important as they ever were.

A third important complication contributing dramatically to "shock" is pulmonary embolism. Since you work in the vicinity of pelvic veins and venous trunks leading from the extremities, your patients would seem to be particularly susceptible to this sort of accident. I am surprised that it does not occur more often than it does. Production and migration of venous thrombi are still poorly understood. For all our study, arguing and attempts at prevention, the incidence and mortality of this complication among surgical patients as a whole remain disturbingly high.

In summary then: "Shock" is not a disease; it is itself a complication. It is not a pathologic entity, but rather the variegated reactions of the intricate machinery of the living body to different sorts of injury or to vital deficiencies. These reactions may give rise to a great variety of symptom complexes. "Shock" is an acute process, a progressive condition and a potentially dangerous one. In "shock" an ounce of prevention is worth a pound of cure. "Shock" in early stages is a relatively simple physiologicopathologic state with borderline decompensation of vital processes. At this stage, rational treatment is comparatively easy and successful. In more advanced stages of "shock" the physiologic responses become increasingly complex and abnormal, irreversible pathologic changes occur, and treatment becomes difficult and less successful.

The main *initiating* causes of shock in colonic surgery would seem to me to be acute blood loss, acute plasma losses, and acute severe fluid and electrolyte imbalances. Each produces characteristic and recognizable syndromes. The important *contributing* causes of "shock" include chronic debility of patients from long-

standing disease, injudicious use of drugs, overuse of intravenous therapy and occurrence of severe postoperative complications.

If "shock" is viewed in physiologic terms and is treated rationally in early stages, it need rarely become a serious problem, or add significantly to the mortality or even the morbidity of modern day surgery. On the other hand, the surgeon who ignores the ever present potential danger of "shock" or who neglects "shock" in its early stages and then treats it injudiciously, surely sooner or later will be plagued and saddened by unnecessary complications and preventable deaths.

Intestinal Antisepsis

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AN acceptable preoperative intestinal antiseptic today should possess certain properties which include: (1) low toxicity and limited absorption from the gastrointestinal tract to maintain its antibacterial activity during the postoperative period of paralytic ileus; (2) rapid, highly bactericidal activity against pathogenic organisms viable in the gastrointestinal tract; (3) prevention of development of overgrowth of pathogenic bacteria or fungi; (4) chemical stability in the presence of the gastrointestinal digestive ferments; (5) activity in the presence of intestinal contents thereby permitting adequate intake of food and liquids; (6) aid mechanical cleansing of the bowel without causing dehydration; (7) non-irritating to the bowel mucosa; (8) will not inhibit tissue growth or interfere with healing; (9) solubility in water, palatability; and (10) restricted use.

It is important that the agent be sparingly absorbed from the gastrointestinal tract and be of low toxicity because of the relatively high concentration of the antibacterial compounds which must be maintained in the bowel if they are to be consistently effective. Even limited absorption of a toxic substance would cause so many untoward reactions in the host as to preclude their use. These substances should act rapidly and be bactericidal so that preoperative preparation need not be protracted. A bactericidal substance will kill the organisms between the folds of the mucous membrane and not require that they be removed mechanically. It is absolutely imperative that the environment of the gastrointestinal tract not be altered by an antibacterial agent so as to permit pathogenic organisms to multiply freely.

THE CHOLERIFORMIC SYNDROME

Probably the greatest single hazard imposed by the administration of an agent which alters the established bacterial flora of the gastrointestinal tract is the danger of permitting pathogenic organisms, yeasts or bacteria, to grow uninhibited in the gut of the host.

The outgrowth of a Micrococcus pyogenes which produces a potent enterotoxin has occurred following the administration of aureomycin, terramycin® and chloromycetin® to give rise to the so-called choleriformic syndrome. This syndrome is characterized by nausea, vomiting, bloody diarrhea, hyperpyrexia, collapse and death usually within four to five days.

Even though this complication is recognized and treatment is instituted early, the mortality rate is high. Erythromycin has been advocated to combat the staphylococcus infection but many of these organisms are now resistant to this antibiotic. Also, the patient is vomiting and oral administration by ordinary means is precluded. If a Miller-Abbott tube can be passed beyond the duodenum, neomycin® can then be administered with some hope of eradicating the offending organisms, the fluids and electrolytes being maintained by parenteral therapy.

The development of a bloody diarrhea is not an infrequent sequence following aureomycin or terramycin therapy. Originally it was considered a consequence of infestation by yeasts, especially monilia. This assumption appeared unlikely because the outgrowth of yeasts, including monilia, following the oral administration of neomycin is as great or greater than that observed after aureomycin or terramycin therapy without a single instance of bloody diarrhea having developed in 750 neomycin-treated cases. It is now considered that the bloody diarrhea is caused by bacterial overgrowth, most frequently proteus or staphylococcus. Should such a situation arise, the patient may well be exposed to a tremendous hazard. Because of the tendency for the predominant outgrowth of resistant bacteria to result when an antibiotic is widely used, an intestinal antiseptic should ideally be restricted to this one use. An antibiotic used in the preoperative preparation of the bowel should most

certainly not be incorporated in a mixture used for the control of ordinary diarrhea.

A compound meeting the foregoing specifications and being stable in the presence of the digestive ferments of the gastrointestinal tract without inhibiting tissue growth would maintain optimal conditions for healing and uncomplicated convalescence. (Table 1.)

TABLE 1
PROPOSED INTESTINAL ANTISEPTICS AND THEIR PRESENT STATUS

Agent	Status	Remarks
Sulfaguanidine®	Discarded	Low activity, too highly absorbed, too toxic
Sulfasuxidine®	Useful	A bacteriostatic agent requiring long administration; limited spectrum
Sulfathalidine®	Useful	Same limitations as sulfasuxidine plus tendency to form tenacious stools
Phthalylsulfacetimide	Doubtful	Irregular absorption, activity inconstant
Streptomycin	Discarded	Rapid development of resistant organisms
Aureomycin (chlortetracycline)	Discarded	Low activity, rapidly absorbed, too toxic, rapid outgrowth of enterotoxic staphylococci
Terramycin (oxytetracycline)	Discarded	Same limitations as aureomycin
Chloromycetin (chloramphenicol)	Discarded	Same limitations as aureomycin plus possible development of blood dyscrasia
Neomycin	Useful	Outgrowth of Aerobacter aerogenes in some geographical areas; yeasts grow out; low absorption; it is not effective against shigella
Neomycin plus sulfathalidine	Best	Yeasts grow out; action rapid; effective throughout period of postoperative paralytic ileus
Neomycin plus terramycin	Doubtful	Enterotoxic staphylococci may grow out

Many other substances and combinations of substances have been proposed as intestinal antiseptics, but they show so little promise that their inclusion cannot be justified.

LOCAL HEALING AND INTESTINAL ANTISEPSIS

It is essential that local healing in the gastrointestinal tract not be retarded by intestinal antisepsis whether by its direct effect upon the tissues of the bowel, by inducing local bleeding through causing vitamin K deficiency or by virtue of permitting overgrowth of pathogenic organisms such as a species of M. pyogenes which elaborates a potent enterotoxin.

It has been demonstrated that sulfasuxidine, sulfathalidine and neomycin do not inhibit local healing but actually favor the process by converting it to one simulating primary tissue repair. Following the administration of these intestinal antiseptics, edema at the suture line is greatly reduced; tissue necrosis does not occur; leukocytic infiltration is minimal; fibroplasia is prompt and the ingrowth of vascular capillaries is not suppressed as is the case when intestinal antiseptics are not employed.

CLINICAL APPLICATION OF INTESTINAL ANTISEPSIS

At present the combination of neomycin and sulfathalidine is the most practical for preoperative intestinal antisepsis. The action is rapid, consistently effective and continues throughout the immediate postoperative period of paralytic ileus and may, if indicated, be maintained indefinitely by renewed administration of the drugs as soon as peristalsis returns.

Patients fall into three categories as to therapeutic technic of antisepsis applicable.

Group 1 consists of individuals without intestinal obstruction to whom purgatives may be given. Preoperative preparation is accomplished in twenty hours scheduled as follows: (1) 60 cc. castor oil, 1.00 gm. neomycin and 1.5 gm. sulfathalidine at 1:00 P.M.; (2) 1.0 gm. neomycin and 1.5 gm. sulfathalidine at 2:00 P.M., 3:00 P.M., 4:00 P.M., 8:00 P.M., 12:00 A.M., 4:00 A.M. and 8:00 A.M.; (3) operation scheduled for 9:00 A.M.

It can be expected that the third stool passed after the castor oil and antibiotic administration, which usually occurs within three hours, will contain only a few organisms or be sterile.

Group 2 consists of individuals with partial intestinal obstruction requiring cautious handling, gradual decompression and more prolonged preparation. Purgation is not permissible. Gastric suction is instituted until decompression is accomplished and gas is passed. Neomycin, 1.0 gm., and sulfathalidine, 1.5 gm., are then given at four-hour intervals for a minimum of twenty-four hours or until optimal mechanical preparation of the gastrointestinal tract is obtained. This period is usually about three days.

Group 3 consists of individuals requiring urgent or emergency surgical treatment demanding undelayed abdominal exploration. Preoperative decompression or measures directed toward the elimination of the bacterial flora cannot be undertaken. Laparotomy is

done as soon as warranted by the general condition of the patient, and antisepsis is accomplished as indicated in the following section of this paper.

USE OF NEOMYCIN IN EMERGENCY ABDOMINAL SURGERY

One of our most serious problems in intra-abdominal surgery is that presented by the necessity of doing an immediate exploratory laparotomy on a patient whose gastrointestinal tract cannot be prepared, either mechanically or with intestinal antiseptics, prior to the procedure. These cases consist of perforation of the bowel due to gunshot injuries, stab wounds, automobile accidents and the like, and also those patients with acute intestinal obstruction with or without necrosis or perforation of bowel, as well as the patient with inflammatory conditions such as Meckel's diverticulitis, acute perforated appendicitis, perforated diverticulitis or perforation of bowel involved in regional enteritis and so forth.

Upon opening an abdomen which is grossly involved with contamination and peritonitis, the excess free fluid is aspirated and the peritoneal cavity is flooded with 200 to 300 cc. of 1 per cent neomycin solution. This quantity of antibiotic will not be sufficient to cause acute toxic symptoms because the major portion will be lost during the course of the exploration and further aspiration. However, the instillation of this highly bactericidal antibiotic will effect destruction of nearly all the organisms contaminating the abdominal cavity within a matter of thirty minutes. Since nearly all of this antibiotic will be removed during the course of the procedure, another 100 cc. of 1 per cent neomycin can safely be instilled just before closing the peritoneal cavity at the end of the operation. The value of this procedure is demonstrated by the fact that feces in 10 per cent suspension will, for all practical purposes, be sterilized by ½ per cent neomycin in the course of thirty minutes while 99 per cent of the organisms are killed within five minutes.

If there are holes in the bowel, these are closed by whatever method is indicated and the lumen of the bowel is then flooded with 1 per cent neomycin. It is frequently desirable to fill both the small bowel and the large bowel with this antibiotic, and quantities as great as 1,000 cc. may be employed. It is introduced through the wall of the bowel using a small needle. No subsequent closure of the needle puncture is necessary. Ordinarily the antibiotic solution is instilled proximally and allowed to travel distally. A warning must be issued at this point should the patient be an infant. The anesthetist must be warned to begin aspiration of the stomach immediately on beginning the instillations, and great care should be exercised to prevent pulmonary aspiration of the solution because a surprisingly small amount of a solution instilled into the ileum of an infant will run back into the stomach and be regurgitated.

Should resection of a segment of bowel be indicated and permissible, that segment is clamped off without preparation of the bowel for definitive anastomosis and the bowel proximal and distal to this area is filled with 1 per cent neomycin solution. One proceeds with the preparation and resection, and after approximately thirty minutes the site of the anastomosis is reclamped and excised in an area which by then will have become essentially sterile. The anastomosis may then proceed using the open type of operation which will afford much more accurate approximation and better preservation of the all-important blood supply.

In the course of the past four years, fifty-two patients have been treated in The University of Texas Medical Branch Hospitals where it has been necessary to close puncture wounds or perform resections in patients who could not be prepared prior to operation. In no instance has peritonitis persisted after operation, have suture lines failed or intra-abdominal abscesses and fecal fistulas developed. Frequently the patient will experience an essentially afebrile postoperative course unless there is an established peritonitis at the time of operation. It is not unusual for a patient to become afebrile within forty-eight hours even though peritonitis was present at operation.

The neomycin which is instilled into the bowel under these circumstances will remain for the duration of the postoperative paralytic ileus and continue to exert its bactericidal effect.

COMMENTS

It has not been the purpose of this presentation to establish intestinal antisepsis as a worth-

while procedure. I believe that the use of intestinal antisepsis over the past fourteen years has now established it rather firmly in our armamentarium of gastrointestinal surgery, especially where the large bowel is involved. It can probably be said that intestinal antisepsis is one of the more important recent factors making gastrointestinal surgery a much safer procedure in the hands of the occasional operator. I do not wish to imply by this statement that it has not been of very definite aid to the well trained surgeon. In my own practice during these fourteen years I have not encountered a single instance of peritonitis due to failure of the suture line or development of a fecal fistula, with one exception. In a one-stage procedure on a child, an abscess developed in the region of a pull-through for megacolon requiring drainage transperineally. In this instance the complication was actually due to a gross technical failure.

At the moment, the combination of neomycin and sulfathalidine gives very satisfactory intestinal antisepsis. However, in view of the past history of antibiotics and the development of bacterial resistance, especially by the staphylococci, it must be anticipated that sooner or later resistant strains of organisms may develop against neomycin if its widespread use in diarrheal mixtures and skin ointments is continued. So far those individuals who have been most interested in observing the possible occurrence of resistant staphylococci have found only rare instances when organisms have been resistant against this antibiotic. It should be appreciated that the therapeutic concentration of neomycin maintained is approximately 5,000 gamma per ml. Nevertheless, the use of neomycin in a diarrheal mixture and then the very likely subsequent use of this substance in the same individual as an intestinal antiseptic, should present a real possible hazard. It certainly should be urged that an antibiotic with such limited use as neomycin should have its therapeutic uses restricted to those of intestinal antisepsis. I know of no group that would be in a better position than the American Proctologic Society to urge the restricted therapeutic use of this substance. Those of you who have observed the rapidity with which a highly virulent staphylococcus can kill through the elaboration of an enterotoxin within the bowel itself will readily appreciate the seriousness of this hazard. Fortunately, it is not of frequent occurrence.

The fact that neomycin or the combination of neomycin and sulfathalidine can be used for rapid preoperative bowel preparation in conjunction with purgation, for more prolonged preparation in the presence of partial obstruction when purgation is undesirable or for use by direct instillation into the lumen of the bowel at the time of emergency operation where any form of preoperative preparation of the bowel is precluded, would establish these antibacterial agents as acceptable intestinal antiseptics. The rapidity of the bactericidal action of neomycin is such that the bacterial flora will have been altered in a period shorter than is required for physiologic and mechanical preparation or decompression of the gastrointestinal tract. The practice of intestinal antisepsis need not increase the length of preoperative preparation and it definitely lessens the morbidity and mortality accompanying intra-abdominal surgical procedures.

Cardiac Arrest and Vascular Complications of Colonic Surgery

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CARDIAC arrest in the operating room is one of the greatest emergencies which can occur in present day surgery. It may happen regardless of how good the patient's preoperative condition may have seemed or how skilled the surgeon or anesthetist may be. During the past few years, however, considerable attention has been called to this problem and both clinical and laboratory studies have been carried out in an effort to improve methods of recognition and treatment.

Stephenson, Reid and Hinton[1] gathered from several countries data on 1,200 cases of cardiac arrest and from them deduced some interesting statistics. Sixty-one per cent were males and 39 per cent were females, although a slightly higher percentage of females undergo surgery than males. Cardiac arrest occurred more often during the first decade of life than in any other age group; the incidence then gradually diminished but rose again as age increased. Hospital data indicated that 14 per cent of the cases of cardiac arrest were encountered outside the operating room. Although cardiac arrest occurred during almost every type of surgical procedure, its incidence was twice as frequent in abdominal surgery as in other fields. This report, as well as numerous others, emphasizes the importance of recognizing and properly dealing with this emergency when it arises.

The successful treatment of cardiac arrest requires immediate recognition that the heart has stopped or slowed appreciably. The pulse and blood pressure are the most obvious diagnostic criteria of cardiac arrest and are usually discovered by the anesthetist, who is in a position to observe these signs closely throughout the operation. When the surgeon has been notified that the pulse and blood pressure are unobtainable, he should immediately try to confirm these findings by palpating an exposed artery. This is a relatively simple matter if he is operating in the abdominal cavity where he may palpate the aorta. If he is not, however, he should try to locate one of the femoral pulses or the aortic pulse through the abdominal wall. There is no time for auscultation or the use of other time-consuming diagnostic methods in verifying cardiac arrest, since the central nervous system will not survive if it has been present for longer than three or four minutes.[2] Stephenson and associates[1] found that in 94 per cent of the successfully and permanently resuscitated cases, cardiac massage was instituted within the first four minutes. Whether or not the heart may be resuscitated is directly dependent upon the rapidity with which the decision is made to start resuscitative measures. If an electrocardiograph is connected to the patient, it may be helpful in determining the onset of cardiac arrest. If in doubt as to whether or not the heart has stopped beating, one should not delay but should open the chest and begin cardiac massage immediately. If cardiac arrest occurs during the course of an operation, the instruments necessary to open the chest are readily available; but, if not, I should like to emphasize that one needs only a scalpel to make the incision into the chest. Sterile gloves are desirable but not necessary. While the surgeon is exposing the heart and initiating massage, the nurse or assistant may obtain a rib spreader and hemostats. If one is operating in the abdomen, it is permissible to split the anterior portion of the diaphragm, insert a hand through it and begin cardiac massage; but the preferred approach is through a separate anterolateral incision in the fourth or fifth left intercostal space. The costal cartilages above and below the incision may be divided adjacent to the sternum to permit more adequate exposure. Time should not be spent counting the ribs to determine such a position. If one makes an incision from the left border of

the sternum, passing beneath the left nipple into the axilla, the fourth or fifth intercostal space will usually be exposed. This incision can be made in only a few seconds and one will not be bothered by excessive bleeding from the wound until cardiac contractions are reinitiated. The pericardium should not be opened initially because of the time consumed in opening it and because, in many instances, the heart will begin contractions again after a very short period of massage through the intact pericardium. On opening the chest, one may find that very feeble and ineffective beats are occurring, that the heart is in complete standstill or that ventricular fibrillation has occurred. In each case cardiac massage should be done in order to supply blood to the central nervous system. It may be helpful to occlude the aorta immediately below the origin of the great vessels with the fingers of the other hand or with a non-crushing clamp, so that the cerebral blood flow will be more effective.

If contractions cannot be reinitiated after one or two minutes of massage, then intracardiac drugs should be given. Atropine 0.4 mg. should be given and 3 to 4 cc. of a 10 per cent solution of calcium chloride, as advocated by Kaye and Blalock,[3] should be injected into the left ventricle. Calcium chloride is an excellent stimulant and is not as prone to produce ventricular fibrillation in the anoxic heart as adrenalin. If cardiac contractions are not reinitiated after injection and further massage, either the calcium chloride may be repeated or two-tenths of 1 cc. of 1 to 1000 adrenalin may be injected into the left ventricular cavity.

If ventricular fibrillation is present, one should try to defibrillate the heart after one or two minutes of massage. Ventricular fibrillation is a condition in which the muscle bundles are contracting irrespective of any rhythmic pattern and has been described as giving the sensation of holding a bag of worms when one holds the heart in his hand. It will not cease spontaneously and can best be arrested by an electric shock defibrillator. Such an apparatus supplies a current through flat electrodes placed on either side of the ventricles and produces uniform contraction of the muscle bundles. One hundred fifteen to 130 volts at 1.5 to 2.5 amperes for varying periods of time has been recommended, but the more recent trend is to give shocks of higher voltages up to 220 or 270 volts for shorter periods, not to exceed one-tenth of a second.[4] The short duration of the shock prevents burning of the heart muscle at the point of contact with the electrode. It may take several shocks before the ventricular fibrillation is abolished. If a defibrillator is not available, procaine or procainamide may be injected directly into the ventricular cavity. In our experience this medication has defibrillated the heart in several instances, but it has the distinct disadvantage of reducing the irritability of the heart to such an extent that reinitiation of contractions by cardiac massage is made more difficult.

There is no general rule for the length of time cardiac massage should be performed. Successful cardiac resuscitation has been reported after more than two hours of massage, and the duration must be determined according to the individual patient. There is some controversy as to the rate at which the heart should be compressed. Johnson and Kirby[5] have shown that cardiac output increases as the rate of cardiac massage increases, and that a rate of 120 contractions a minute produces an optimal blood flow. From a practical standpoint, however, a slower rate is usually necessary, and we attempt to maintain the rate of seventy to eighty contractions per minute. One should cease massage if one feels effective cardiac contractions begin or should pause at intervals of one or two minutes to see if spontaneous contractions are occurring.

I have been speaking primarily about the role of the surgeon in cardiac arrest, however, the anesthetist has an equally important role. It is a sine qua non that from the moment cardiac arrest is suspected all anesthetic agents should be suspended and 100 per cent oxygen should be delivered to the lungs under positive pressure. An endotracheal airway is preferable if it is not too difficult to achieve, but before it is attempted positive pressure oxygen should be given by mask for a short period. Blood should be administered rapidly, preferably by interarterial transfusion, to increase the circulating blood volume. However, in most institutions interarterial transfusions are not routinely used and the time lost in setting up such a system after cardiac arrest has occurred reduces its value. Whether or not the original operative procedure should be continued after the reinitiation of contractions has taken place depends on the extent to which the operation has progressed. In the vast majority of in-

stances, however, the procedure should be concluded quickly because of the possibility that cardiac arrest might recur.

The question arises as to why cardiac arrest occurs and why its incidence shows an absolute increase. One might attribute this increase to the more complex procedures now being performed, particularly in thoracic and cardiovascular surgery, although as we mentioned earlier, the greatest number occur in abdominal surgery. Another contributing factor is that more poor risk patients are undergoing operations today because of advances in technics of anesthesia. In this regard the multiplicity of anesthetic agents and drugs administered by the anesthetist may well be an important factor. It has been shown that anoxia greatly increases susceptibility to cardiac arrest, and more recent work implicates hypercapnia as possibly being more important.

Vascular complications associated with surgery of the colon may be classified as those which occur in the operative area and those which occur in other areas subsequent to the operative procedure. Of those associated with the operation itself, hemorrhage is probably the most frequent, following either right or left colon resection, especially in the patient having a bleeding tendency, although no gross blood dyscrasia may be present. Patients who have had massive transfusions of citrated stored blood may have excessive oozing because of a platelet deficiency in the transfused blood. Such oozing may require a direct transfusion of fresh whole blood which in most instances produces dramatic arrest of the oozing. Bleeding may result from a loosening of a ligature, particularly in the obese individual, or may occur at the site of the anastomosis. Late hemorrhage may occur, often as a result of an infection which causes blood vessels to slough. Usually hemorrhage is not difficult to diagnose when one observes the patient's red blood count and hemoglobin, the pulse and blood pressure, and any changes in the abdominal signs.

Gangrene of the bowel is a more elusive problem, primarily because of the insidiousness of its onset and the difficulty encountered in its diagnosis. Probably the most frequent cause of gangrene is interference with collateral circulation, which may be difficult to ascertain at the time of operation, especially in the presence of large, invasive tumor masses. Thrombosis of mesenteric vessels may result from operative trauma or infection. In the older patient, traction on the mesentery may produce a rupture of the intima of a major mesenteric artery, especially if atheromatous changes have occurred, with subsequent thrombosis and compromise of the circulation to a colon segment.

Trauma to the femoral and iliac veins may contribute to the subsequent development of thrombotic phenomena in these vessels. Venous thrombosis most often begins in the lower extremities as a result of stasis, however, and extreme care should be used to prevent its onset. The prevention of such postoperative vascular complications as phlebothrombosis and thrombophlebitis should begin on the operating table. One should not bind the lower extremities tightly to the table, nor should one place large sand bags under the popliteal fossae. If the patient is in stirrups, one should be sure that excessive pressure is not exerted in the popliteal fossae. It is desirable to measure the circumference of the lower extremities before and after the operation, especially on the patient who is placed in stirrups, and these patients should be observed closely to determine the onset of swelling or coldness of an extremity or development of a positive Homans' sign. The importance of passive and active exercises and of early ambulation cannot be overemphasized.

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DISCUSSION OF SYMPOSIUM ON COLONIC SURGERY

Alton Ochsner (New Orleans, La.): I am grateful for the choice of men who have served on the panel because it makes my job as a discussant very easy. The one thing that impressed me is that each of the authors emphasized the importance of prophylaxis of complications. If we can prevent something from happening, it is very much easier.

Dr. Poth has emphasized so well the importance of the preparation of the bowel and the need for us physicians not to abuse neomycin. It is a valuable drug, but its use should be limited to conditions in which it is actually necessary. This has already been alluded to by Dr. Price.

I think as physicians we have been guilty of being stampeded into doing many things by our patients, because if patients demand something or want some type of therapy, we will give it to them, but by doing so we are likely to produce a sensitivity to the drug or to make an organism resistant to the drug. I hope we will all accept the admonition which Dr. Poth gave us to refrain from the use of neomycin when it is actually not needed.

Dr. Price has emphasized the importance of prevention of shock, and he has done it well. It is important, as he emphasized, to individualize each case. It would be a lot easier if we had a formula to follow, but unfortunately medicine cannot be carried on according to formulas because our patients are human beings. They are biologic subjects and biologic phenomena do not behave according to a formula. It is extremely important to individualize each case and prevent the complications which Dr. Price discussed.

I should like to emphasize again, as far as Dr. Muller's contribution is concerned, the importance of prophylaxis. He has succinctly told the possible reasons for the increase in cardiac arrest. It is increasing not only because cardiac patients and poorer risk patients are being operated upon, but also because we are using multiple preanesthetic drugs and anesthetic agents which were not used previously. We are sensitizing the cardiac musculature of patients as we never did before.

In my paper on colonic surgery I paid tribute to the modern anesthesiologist and again I want to emphasize their value, but too frequently they use a whole gamut of drugs with real detriment to the patient. We did not see many cardiac arrests previously because patients were treated very simply. Preoperatively they were given morphine, and a liberal dose of atropine and ether was the anesthetic agent of choice. That combination is still good. We seldom need many of the fancy new drugs, and I am sure that pentothal and the various relaxant drugs predispose to cardiac arrest. Unfortunately, many times patients are not properly sedated before being brought to the operating room. They are agitated and this causes an increase in the output of adrenalin which in turn sensitizes the cardiac musculature.

I am grateful to both Dr. Price and Dr. Muller for mentioning venous thrombosis, because this is a subject in which I have been interested for many years.

Venous thrombosis has become a complication of increasing importance postoperatively. The previously encountered causes of death following operations, shock and infection have been largely eliminated. Venous thrombosis, therefore, has become relatively more important and also is actually increasing. What the increased venous thrombosis is due to, I don't know. At one time I thought that it might be due to the almost ubiquitous use of antibiotics, and I think that this still may be a factor. Regardless of the cause, the fact remains that venous thrombosis is increasing. Only within the past two years have we been able to effect a decrease in its incidence. Whether this is due to the fact that we are just going through a cyclic change which is so frequently true of venous thrombosis or not, I don't know, but I think it is due to therapy. It is of importance to differentiate between two types of venous thrombosis, because these two types are as different as night is from day, although, I know some will not agree with me. The two types of venous thrombosis are phlebothrombosis and thrombophlebitis. The former is a coagulation thrombus within the vein unassociated with vein wall inflammation, and for this reason the clot is not attached to the vein wall and can be removed easily. In the latter condition, thrombophlebitis, the clot is secondary to an inflammation of the vein wall and is firmly attached. It is true phlegmasia alba dolens. There is usually no difficulty in making a diagnosis of thrombophlebitis, because the patient has pain, fever and swelling of the extremity. Phlebothrombosis, on the other hand, is extremely difficult to diagnose. There are few or no manifestations and, in fact, in 40 per cent of our cases of fatal pulmonary embolus, almost invariably associated with phlebothrombosis, there was no antecedent evidence of venous thrombosis. In thrombophlebitis, unless adequate therapy is instituted early, edema persists for a long time and other postphelbitic sequelae develop. These patients

are seen in our surgical outpatient clinics for months and years and, frequently, for the rest of their lives. Treatment of thrombophlebitis consists of vasodilatation secured by blocking the regional sympathetic ganglia with procaine. If this is done relatively early, complete restitution is obtained quickly.

On the other hand, phlebothrombosis is not due to inflammation of the vessel wall but is due to two factors; one is an increased coagulability of the blood which is due to an increased clotting tendency secondary to tissue injury and the other is circulatory stasis. Apparently it doesn't make any difference what causes the tissue injury, whether it is due to accidental trauma, operative trauma, invasion of tissue by neoplastic disease or infection, but if there is tissue injury, which undoubtedly is a protective phenomenon, there are changes in the blood constituents increasing the blood coagulability.

Most phlebothromboses occur in the veins of the lower extremities because of circulatory stasis. The coagulation thrombus in phlebothrombosis is a red thrombus and is similar to the clot that occurs in a test tube. If blood is withdrawn from the body, and placed in a test tube and allowed to clot and then the test tube is turned upside down, the clot remains attached for a short period of time and then becomes detached. That is exactly what happens in a patient with phlebothrombosis. Phlebothrombosis occurs generally without any symptoms. The patient may have a sense of impending disaster and have an elevation of pulse rate out of proportion to anything else. They may have, as Dr. Muller mentioned, tenderness in the calves of the legs or they may have no evidence of venous thrombosis at all.

The first manifestation of phlebothrombosis is frequently a pulmonary infarction. Fortunate is the individual with phlebothrombosis who gets a non-fatal pulmonary infarct, because the diagnosis of phlebothrombosis is relatively easy and the involved vein can be ligated on the cardiac side of the thrombus.

There are many who will not agree with this concept. There are many who believe that one should give anticoagulants. However, I do not believe that in phlebothrombosis one can prevent detachment of the clot by administration of anticoagulants. It is true that one can prevent the propagation of an existing thrombus or the formation of more thrombi, but one cannot prevent the detachment of the original clot. I fear that much harm is done by administration of anticoagulants because the physician is lulled into a false sense of security that he is treating his patient adequately, whereas he does not prevent the detachment of the non-adherent clot.

If anticoagulants are to be used, they should be employed prophylactically, but the usually employed anticoagulants, dicumarol, heparin and tromexan and some of the newer ones are far too dangerous to use routinely.

About three years ago, based upon experimental observations, we suggested the prophylactic use of alphatocopherol and calcium, because *in vitro* the combination of alphatocopherol and calcium exerts an antithrombic activity.

We were disappointed after a period of time to find that others had not been able to substantiate our *in vitro* observations. So, about two years ago at the Charity Hospital we discontinued the use of alphatocopherol and calcium on the Tulane Service for a period of six months. However, we continued its use at the Foundation Hospital. The Staffs are approximately the same at the two hospitals, the types of cases are approximately the same, and the period of time was the same. During the six-month period of observation the incidence of pulmonary embolus at the Charity Hospital was high, whereas at the Foundation Hospital it was extremely low. We are definitely of the opinion that the postoperative administration of alphatocopherol and calcium exert an antithrombic effect and yet are safe to use routinely because they do not produce a hemorrhagic tendency.

WENDELL GREEN (Toledo, Ohio): Several questions have been directed to the panel. We will start with one for Dr. Muller. Have you known any patient who has recovered following cardiac standstill when the electrocardiogram has demonstrated no action currents?

WILLIAM H. MULLER (Los Angeles, Calif.): That is a very difficult question to answer because I do not believe it is too well documented. In our own experience with patients who have undergone cardiac surgery cardiac contractions have been reinitiated after virtually no action currents or only an occasional pip on the oscilloscope. In at least one or two incidences these patients have been permanently resuscitated.

PHILIP B. PRICE (Salt Lake City, Utah): Several questions have come up about circulating blood volume and the methods used.

In general there are two methods or two classes of methods employed. Perhaps the simplest of these is the dye method in which a known quantity of dye, usually Evans blue, or T-1824 is injected. The concentration of dye in samples of plasma is then measured with a photoelectric colorimeter. This method measures plasma volume not total blood volume.

The other method uses radioisotopes and can be used to measure either the plasma volume, by tagging materials injected such as protein, or the red cells, by tagging some red cells and injecting them. Either method, however, is rather tricky.

I think the best that one can hope for from any sort of a quick bedside method would be to admit that the results are not very accurate but they do give a fair indication of plasma volume or blood volume, and sometimes this is very useful.

Of the two methods the dye method is less expensive and takes less training of personnel. The radioisotope method, once set up, can be done with less trouble. The percentage of error depends on the person who is doing the test.

In the laboratory, working with dogs, I came to the conclusion that we could reduce the error in plasma volume determinations to something less than 5 per cent. With human subjects the error would be much greater than that in the best of hands, and some estimates have run as high as 30 per cent.

Another question is whether in replacement of chronic blood deficit the patient's age is a factor.

I think the age is something of a factor because, as I have tried to point out, in any long extended blood deficit what one is trying to do is not merely to replace blood or blood elements in the vascular bed itself but in the body generally. That is harder to do. It takes longer in an aged person. In acute hemorrhage I am not sure that age makes much difference.

Here is a question that I cannot answer. Maybe some of the others on the panel can. "Do you think that hibernation as advocated by French authors will contribute to the prevention of surgical shock?"

My only experience with that principle has been local refrigeration, say of a gangrenous extremity, and in that sort of case I think shock can be reduced if the method is properly carried out.

"What are your ideas of the relaxants and prolonged anesthesia, and the newer idea that the surgeon has all the time in the world, so to speak?"

I think one has to hit a middle-of-the-road policy here. Greatly prolonged anesthesia and greatly prolonged time on the operating table is in itself a strain on the patient's body machinery and the function of his vital organs. On the other hand, if you push ahead and perform operations quickly but carelessly, I think, you can err as much or more on the other side. After all, the slow operation isn't really a new idea. I can remember that my old chief, Dr. Halsted, used to come in for a great deal of criticism because he and his students often took a long time to operate, and yet I rarely saw a case of a badly shocked patient in the prolonged breast operations, for example, which he did. I think that was the result of his very careful work.

I have been asked to comment on preexistent adrenalin insufficiency as a possible cause of shock during and shortly following operation.

Well, of course, where there is a real adrenal insufficiency, let us say as in Addison's disease, shock can occur very readily. Minor degrees of adrenal insufficiency would be fairly difficult to spot.

"Is pain itself a factor in producing shock or is the shock produced by factors causing the pain?"

Both can occur. Pain itself can be a contributing factor in the production of shock. Emotion associated with pain can produce extra adrenalin and that in turn can certainly contribute to disturbed physiology and perhaps to shock.

DR. OCHSNER: My first question is: Do you consider that there is always intravenous clotting in thromboplebitis?

Yes, I do. I think that the patient with phlebothrombosis may subsequently get inflammation if he is fortunate enough in not having his clot become detached and doesn't develop an infarction.

Next is the question of injection of alpha-tocopheral and calcium in postoperative care.

There is an intramuscular preparation put out by Warren-Teed which is called epsilan.® Please don't misunderstand me. I do not have

stock in Warren-Teed and I am not trying to sell any product but we use this alphatocopheral product because it is the only one I know of in which the alpha fraction has been acid and which is the only one that exerts an antithrombic effect. That I know and unless you know what the alpha fraction is it is not worth using.

When using the intramuscular form which is epsilan phosphate, one administers 100 I.U. every eight hours; with the oral form 200 mg. every eight hours and the 10 cc. of gluconate is given intravenously every twenty-four hours.

The calcium is apparently necessary for the alphatocopheral to exert its thrombic effect, at least in phlebitis.

I should like to tell about a patient, a famous individual in one of our very well known university centers, who was about to undergo an operative procedure. As he was being put to sleep cardiac standstill developed. His chest was opened up and his heart was massaged for five and one-half hours, and during the course of the time the ventricle was punctured twice. It was sutured and at the end of the five and one-half hours cardiac mechanism was reestablished. The patient was perfectly well for a number of months but subsequently died of coronary disease. This shows, however, what can be accomplished and that one should not give up even after you put your thumb through the ventricle.

EDGAR J. POTH (Galveston, Tex.): The question I've been asked is whether neomycin 1 per cent suspension is commercially available and how it must be prepared in the hospital for this purpose.

Neomycin is very highly soluble and would not be in suspension but rather in solution. A lyophylized ampule which contains 5 gm. is available. I understand other products are going to be made available very shortly in the form of sterile powder. Neomycin itself will withstand boiling. You can take any tablets, dissolve, strain off the carrier in the tablets and boil.

Why do you prefer sulfathaladine or sulfasuxidine?

I presume this is in combination with neomycin. I do not prefer sulfasuxidine over sulfathaladine with neomycin. Sulfathaladine is effective in about half of the dose as sulfasuxidine and, therefore, a smaller dose will be effective in the presence of the watery stool which is produced by neomycin. You are not concerned about formation of a tenaceous stool by sulfathaladine when combined with neomycin.

What antibiotic do you recommend for perianal inflammation without suppuration?

Of course, if this is a cellulitis, I would say the same indications as anywhere else, systemic administration. It certainly would not be neomycin. If it is a matter of wanting to treat the surface, then you can use neomycin and sulfathaladine by mouth. In order to control the diarrhea administer paregoric. It is quite useful for certain types of open wounds perianally. Otherwise the same antibiotic would be used for cellulitis as elsewhere in the body depending upon the indications.

Is any antibiotic advantageous in diarrhea mixtures?

I would say absolutely no, and there is no indication for using any antibiotic at any time in connection with non-specific diarrheas.

I have been asked to discuss the indication and hazard for parenteral administration of neomycin.

There are very few indications for the parenteral administration of neomycin. It has been used, of course, in patients with tuberculosis and is still being used in that connection. It has the disadvantages of otic nerve toxicity which is greater than streptomycin. We have given neomycin parenterally rather than topically in the peritoneal cavity.

Where we are giving it intraperitoneally it is really for topical application rather than parenteral administration. We are not using it for systemic effect but for its effect on the peritoneum for short duration. You must not give too much. Just exactly what the maximum tolerated dose in the human is I wouldn't know. It acts through respiratory arrest in acute toxicity.

Do you perform proximal enterostomy following primary anastomosis?

No, I don't because I am inclined to consider a condition which would indicate the need for proximal enterostomy to be a contraindication for a primary anastomosis.

DR. MULLER: My next question is: In connection with cardiac arrests Dr. Hanton et al. stated that scopolamine does not have the protective properties of atropine as a preoperative medication. Do you agree with this?

Yes, I think that we agree with it. One de-

sires to block the vagus nerve to prevent a vago-vagal reflex and we think that atropine probably does this better than scopolamine. The next question is:

Is cardiac arrest as frequent under local or regional block anesthesia as under inhalation anesthesia?

No, it certainly is not. The type of reaction which one gets following a local or regional anesthetic is a different one, and is usually treated with one of the barbiturates intravenously. It is most often a type of reaction involving spasm of the smooth muscle particularly in the tracheobronchial tree. The usual local anesthetic does not interfere with the respiratory exchange and therefore the Ph and electrolyte system are not upset and cardiac arrest is less likely to develop.

DR. GREEN: I want to express our appreciation to all the gentlemen who have participated in this symposium. I am sure we all hope that we will never encounter a gross contamination in the abdominal cavity, nor will ever have a patient go into shock or have cardiac arrest. But I am sure that after the principles that have been reemphasized herein, we certainly will be in a better position to handle any of those conditions.

Pathogenesis of Common Anorectal Infections

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THE common anorectal infections or inflammatory diseases comprise hemorrhoids, anal fissure, anal abscess, anal fistula and anal contracture. The chief etiologic agent in these entities is anal infection.

What is meant by anal infection? This has reference to those events occurring in the development of anorectal inflammatory disease. The principal anatomic structures involved are the anal crypts, the anal ducts and glands, and the adjacent blood vessels and lymphatics.[1]

The historic account of anal ducts and glands is interesting, somewhat amazing and involves several men well known as members of this Society.

According to Coleman,[2] Winslow (1733) and Haller (1754) noted the probable etiologic significance of anal crypts in relation to anal fistula. Coleman found that Chiari in 1878 was the first to describe anal ducts and glands. Chiari's work was confirmed by Herrmann and Desfosses in 1880.[3] My impression, until the recent studies of Coleman, was that Herrmann and Desfosses were the first investigators to describe anal ducts and glands in the human.

Johnson's[4] monumental study of the development of the human rectum, including the anal glands, was published in 1914.

Pennington[5] wrote in 1917: "That an important anatomic fact could in this scientific age, be disregarded or lost sight of for many years, after being placed on record, and then, without knowledge of its former discovery, be rediscovered and found to be of great importance as a disease-producing factor, is almost unbelievable. However, this anatomic finding when resurrected, proves to be a vital etiologic factor in the causation of fistula and other diseases of the rectum with their metastatic sequelae."

In 1933 Tucker and Hellwig[6] presented their histopathologic observations pertaining to the anal ducts and glands, together with an excellent comparative anatomic study, and discussed the pathogenesis of anorectal inflammatory disease. This work was confirmed by Pope and Gunn.[6] I believe that the work of these four men constitutes another "rediscovery" and that it has stimulated the present day interest in anal ducts and glands.

More recent contributions to our knowledge of anal ducts and glands have been made by Scarborough[7] and by Hill, Shryock and ReBell.[8] Anson, Coleman and I have engaged in a restudy of anal glands and ducts. This work is at present being prepared for publication. A part of it will serve here to illustrate the salient anatomic features of these structures.

STAGE I OF ANAL INFECTION

During the passage of stool through the anal canal there is excellent opportunity for infectious material to be caught or trapped in one or more anal crypts and then directed into the anal ducts and glands. I believe that neither trauma nor so-called cryptitis is essential to the initiation of anal infection. The anal crypts serve merely as funnels through which infectious material from the gut is guided into the anal ducts and glands. Thus are provided portals of entry into the perianal and in some instances the perirectal tissues without the intervention of trauma to anal skin or to rectal mucous membrane.

It must be remembered at this point that liquid material can more easily gain entrance to the anal crypts and their attached ducts than can material from a fairly dry and well formed stool. This is a point of practical importance in the proper understanding of the anorectal sequelae of diarrheal diseases and of the vicious cycle evolved from the abuse of mineral oil and/or laxatives. Further reference to this problem will be made subsequently.

It seems logical to assume that very little edema of the tissues surrounding the opening of an anal duct would be necessary to cause occlusion of the duct, with resultant retention of infectious material within the duct and within any acinous structures attached to the involved duct.

Attention has already been directed to the round cell infiltration in the tissues immediately subjacent to the openings of the anal ducts and in the periglandular tissues. I believe that this finding is evidence of chronic inflammation.

STAGE II OF ANAL INFECTION

The second stage of anal infection is that of invasion of the perianal and/or perirectal tissues. This can occur directly as a result of breaks in the continuity of the gland-duct structure, possibly from overdistention, or indirectly by way of the blood vessels and especially by way of the lymphatics.

Johnson showed that lymphoid tissue is laid down around the anal glands and ducts at an early period in embryonic life. Buie[9] believes that this early and intimate relationship between lymphatics and glandular structures is a significant factor in the development and spread of anorectal infections.

The arbitrary division of anal infection into stages is, of course, a didactic maneuver. Stages I and II are common to all the anorectal inflammatory diseases. Stage III is best discussed for each of these entities.

STAGE III OF ANAL INFECTION

Hemorrhoidal Disease. On microscopic study of anal ducts and glands it is seen that frequently they lie in close association with the component parts of the hemorrhoidal plexus. Infectious material having gained entrance to the perianal and perirectal tissues, the normally thin-walled veins of the hemorrhoidal plexus are subject to periphlebitis, endophlebitis and thus further thinning of their walls, with resultant dilation in varying degrees. The third stage of anal infection in hemorrhoidal disease varies from a chronic inflammatory reaction which is seen only on microscopic study of the tissues, to an acute reaction characterized by edema, multiple thromboses, gangrene, sloughing and ulceration easily recognized clinically.

Anal Fissure. Should infection be guided superficially in the subcutaneous tissues of the anal wall, most commonly the posterior wall, dissolution of the overlying anal skin may follow. The resultant lesion is an anal ulcer or anal fissure. The failure of such a lesion to heal spontaneously or to respond to any treatment short of adequate surgery is due to the inexhaustible supply of infection-laden material being funneled into the tissues underlying the fissure.

Anal Abscess and Anal Fistula. Here again, infectious material having gained entrance into perianal or perirectal tissues, conditions may favor the development of an abscess. The vestigial anal ducts and glands are located in the superficial subcutaneous connective tissue. Occasionally, however, the glandular elements may invade superficial fascicles of sphincteric musculature. Also, on histologic study a communication between an anal gland and a crypt of Lieberkühn immediately proximal to the dentate line may be seen. These and other anatomic variations, plus such variable factors as virulence of the invading organisms and resistance of the tissues of the host, are probably responsible for the clinical course and manifestations of the inflammatory process.

The abscess can involve one or several of the surgical spaces about the anorectum. The acute process soon acquires chronicity by one of three methods: (1) prompt incision and drainage by the surgeon, (2) spontaneous rupture, or (3) partial drainage spontaneously through the anal crypt of origin. The first two methods result in the formation of an anal fistula. The third results in a chronic anal sinus.

Also of interest in the pathogenesis of anal fistula is the probable role of the lymphatics explaining, in my opinion, Goodsall's rule.

Anal Contracture. Anal contracture which is not the result of injury or of a surgical procedure and which is not congenital in origin can be explained on the basis of anal infection. If for any reason (commonly the abuse of mineral oil and/or laxatives) the anal canal should be subjected to the frequent passage of loose or watery stools over a long period of time, scar tissue is likely to develop in the perianal subcutaneous tissue as a defense against anal infection. Such scar tissue is usually circumferential in distribution. Contracture of this scar results in narrowing of the anal canal. When the individual realizes that he is having some difficulty in evacuating the stool, he may decide to increase the dosage of mineral oil

and/or laxative, thus facilitating the mechanism of anal infection. More scar tissue is deposited, resulting in additional anal contracture. Accordingly, a vicious cycle is established. This type of anal contracture is yet another manifestation of anal infection. Careful endoscopic study of the patient may reveal melanosis indicative of prolonged use of cascara or of a mixture containing cascara.

Combinations of Anal Infection. An additional matter of practical importance pertains to the realization that anal infection can manifest itself in more than one form in one patient.

CONCLUSION

It is my opinion that the "basic science" approach to an appreciation of anal infection and of its role in the pathogenesis of ordinary anorectal inflammatory disease is sound. Such knowledge is essential to an understanding of the objectives of definitive anorectal surgical procedures.

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Comments on the Treatment of Lesions Resulting from Anal Infections

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IN recent years proctologists have benefited through the use of new and effective drugs as adjuncts to the surgical treatment of their patients. When penicillin first came into use it was found that the coliform organisms secreted a resisting factor termed penicillinase which rendered the drug ineffective in the dosage which was then prescribed. We now know that this barrier can be passed if penicillin is given in definitely higher concentration than that required for other organisms. This concentration cannot be properly maintained, however, by even large amounts of the depository type of drug but can be secured only by frequent administration of soluble penicillin. In eleven recent bacterial antibiotic sensitivity tests done on the Proctologic Service at Baylor Hospital from material from anorectal abscesses, Entamoeba coli was the predominant organism in eight and proteus was the predominant or accessory factor in three. The bacteria were found resistant to penicillin in every case and to streptomycin in eight. Therefore, it is evident that penicillin is not the drug of choice in abscesses due primarily to coliform organisms. Evidence at hand would suggest that chloromycetin® is one of the several more effective agents. In this connection work now being done by Haberman[1] and others suggests that sensitivity testing of bacteria will have a large place in the future in the proper treatment of anorectal infections. It is now possible through a fairly simple procedure to determine which of the available antibiotics will be effective in any given bacterial infection. Among the other investigated organisms important to proctologists, hemolytic streptococcus has been found to be sensitive to penicillin as well as aureomycin in ten of eleven strains, whereas Streptococcus viridans was affected by penicillin in only four of forty-six strains tested. Eighty-six strains of staphylococcus have been tested as to their sensitivity, and in this group only thirty-seven were affected by penicillin, whereas bacitracin was lethal in seventy-five instances. As this type of laboratory procedure becomes available generally we will be in a position to select the most effective antibiotic in each given case, which should have a more definite effect in reducing morbidity.

Clinical experience has demonstrated, however, that the use of antibiotics will neither abort an abscess nor prevent the formation of a fistula, although it will slow the process and change the clinical picture, at times sufficiently to confuse the diagnosis or give the clinician a sense of false security. The explanation for this paradox lies in the fact that in a beginning abscess, with its encircling pyogenic membrane and its poor local circulation, it is impossible for all the bacteria present to be destroyed or benumbed; secondly, the local reaction of body tissues to the injury caused by the presence of bacteria, once initiated, continues to its customary termination. My own experience has been that when such a lesion is explored after the use of antibiotics, cavity formation, granulation and necrotic tissue are still present. Therefore, it is apparent that our present antibiotics are helpful adjuncts to surgery but should not be substituted for the drainage of an abscess or the eradication of a fistulous tract.

The use of appropriate drugs has also been very helpful in connection with tuberculous infections about the anus. Surgical therapy for tubercular abscesses and fistulas has, of course, changed greatly during the past quarter of a century and surgeons have become increasingly bold in their attack upon these conditions in this locality, as they have in the thoracic cavity. We learned some years ago that abscesses could be drained promptly even though occurring in tubercular individuals, and that if a coincident pulmonary lesion is quiescent and fibrotic, a tubercular fistula can be expected to heal after surgery although the period required for healing is more prolonged. Martin[2] and others now believe that the healing process

can be expedited by the administration of appropriate medication before and after fistula surgery. In 1950 Martin reported healing of tubercular ulcers and non-fistulous sinuses in patients on a medical regimen consisting of the intramuscular injection of 0.5 gm. of streptomycin divided into two daily doses for varying periods. In a few cases dihydrostreptomycin was substituted. Because all strains of the tubercle bacillus are not equally influenced by streptomycin, at present Martin[3] is using with even more favorable results a combination of 1.0 gm. of intramuscularly administered streptomycin only twice weekly, supplemented by para-aminosalicylic acid given orally in 4.0 gm. doses three times daily. This medication is given for several weeks before fistulectomy and continued until the wound heals.

Even during modern times one deterrent to successful operative treatment of anal fistulas has continued to be timidity on the part of the surgeon. Since the time of Hippocrates some surgeons have feared to sever even the subcutaneous sphincter because of the possibility of causing incontinence, lacking that intrepidity of mind which Aurelius Celsus (42 B.C.) described as an essential qualification in a surgeon. Both Paulus Aegineta (600 A.D.) and John of Arderne,[4] who lived five centuries ago, advised the timorous to treat the tract with a ligature in the manner of Hippocrates, but recommended to other surgeons that a probe be introduced into the fistula and all the intervening tissues be divided with the knife.

Those who have failed to follow their advice have surely been conscientious in their action, probably adhering to the old aphorism, "Physician, first, do no harm," and have had our understanding sympathy and patient instruction, which is bearing fruit. But barely had this desired goal been reached and this attitude of understandable fearfulness overcome in many surgeons, when some of my most esteemed colleagues, apparently impatient of time limitations, began to urge an impetuous frontal attack upon the problem of fistula surgery which would swing the pendulum to the opposite extreme—advocating major anal surgery including fistulotomy in the presence of acute suppurative perianal infection.

The surgical removal of fistulas and sinuses should be both complete and considerate. It should be complete because otherwise infected tissue, septic anal ducts and glands draining into primary crypt openings and even tubercular or malignant lesions may be unrecognized or left in place. It should be considerate of the anatomic factors involved, the viability and healing ability of body tissues concerned and the patient's continence.

In twenty-five individuals with squamous cancer of the anus who have been under my care, the lesion had apparently originated in fistulas in three and an anal ulcer in one. In addition, fistulas and coincident adenomatous cancers of the anal canal have been found in fourteen instances. Whether in any given instance the cancer is one implanted on a previously benign lesion or, as frequently occurs, the tract is a complication of a malignant process, is of only academic importance for the purpose of this discussion. Complete excision of inflammatory anal lesions is preferable because of the possibility of the presence of a neoplasm. Only complete removal of the lesion can assure definite knowledge of its true nature. This is only one of the considerations which has caused many of us to substitute fistulectomy for fistulotomy and the block excision of anal ulcers for dilatation or incision.

I have no quarrel with the advice that a perianal abscess be drained at the earliest possible moment, a procedure urged by Allingham[5] in the last century. Only in this way can we halt the burrowing process and the destruction of essential anatomic structures. On the other hand, we know now that a fistula is, in effect, the origin and not the end of the process. The extensive drainage formerly advocated, combined with removal of large amounts of overlying skin, will not obviate the fistula and, therefore, serves no real purpose. Essential drainage can usually be established as an office procedure and the intervening period before definitive surgery for the fistula can be abbreviated by the judicious use of the indicated drugs. In the past we have found that incontinence following fistula operations has been produced chiefly by prolonged separation of the wound by packing, by the interposition in the wound of nearby hemorrhoids and even by the presence of redundant anal epithelium interposing in the same way and preventing rapid regeneration of necessary granulation tissue.[6] I predict that if the profession indiscriminately adopts the plan of simultaneous drainage of acute abscesses and fistulotomy for the causative fistula, regardless of its location

on the anal perimeter or the depth of the tract, a new factor in the causation of anal incontinence will have been introduced.

SUMMARY

Antibiotics may be expected to reduce pain and morbidity due to anorectal infections. Bacterial sensitivity tests will indicate the specific drug effective in each instance.

The routine use of penicillin or penicillin-streptomycin combinations is not commended, particularly when Entamoeba coli is the predominant organism.

Antibiotics will retard, possibly mask the presence of an abscess or fistula, but serve only as adjuncts to essential surgical procedures.

The use of streptomycin combined with other drugs is advisable in connection with surgery for tubercular fistulas and may heal tubercular anal ulcers and sinuses without surgery.

Timidity on the part of the surgeon has been a prime factor in the failure of cure following insufficient surgery for anal fistulas. A present trend, however, would go to the opposite extreme and suggest radical surgery in the presence of acute pyogenic infection leading, if accepted generally, to incomplete eradication of fistulas, the substitution of fistulotomy for fistulectomy and possibly to an increase in the incidence of postoperative incontinence.

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Treatment of Incontinence and Other Abnormalities Following Infections of the Anal and Rectal Regions

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THE most frequent sequelae of anal and rectal infections which require corrective surgery are incontinence or stricture or a combination of the two. Incontinence is most frequently due to operation for the various types of abscess and fistula. Stricture is more likely to result from infectious processes within the rectum such as lymphopathia venerea, syphilis, ulceration due to ulcerative colitis or any other severe acute or chronic ulcerative process. The infection destroys tissue and the healing processes produce fibrous tissue. The destruction and repair of infection together with the contracture of the fibrous tissue eventually leads to a stricture of the rectum or colon. If the disease is low enough to involve the sphincteric mechanism in the inflammatory process a combination of incontinence and stricture result.

For purposes of discussion, strictures have been termed annular or tubular. The genesis of both types is the same. The length of the segment involved is the criterion for this classification. Arbitrarily, an annular stricture is considered to be one which is 2 cm. or less in length. A tubular stricture is longer than 2 cm. Many types of treatment of rectal strictures have been described in the literature and the very fact that there are many methods of treatment is evidence that there is not any one perfect method of treating strictures. In the annular type of stricture continued gentle dilatation will often give adequate relief but if the dilatations are discontinued contraction almost uniformly recurs. For this reason, unless there is some specific contraindication, surgery is usually the preferable method of treatment.

Some strictures below the peritoneal reflection, especially the annular type, can be treated with multiple radial incisions through the constricted area. If this procedure is used, it is imperative that the scar tissue be completely divided and dilatations done until healing is complete. If for some reason this type of surgery is not feasible and for treatment of longer strictures, resection is the treatment of choice. In treating tubular strictures in which the sphincter mechanism is not involved one of the so-called "pull-through" type operations is usually the procedure of choice. An end-to-end anastomosis is equally effective but with strictures lying below the peritoneal reflection this operation is technically more difficult than the "pull-through" type and carries a greater risk for the patient.

In cases in which the infectious process has greatly impaired or destroyed the sphincter mechanism and both strictures and incontinence are present, the most satisfactory procedure is a permanent colostomy with or without an abdominal-perineal resection. The strictured immobilized outlet is in reality a perineal colostomy on which no suitable appliance can be worn. A well managed abdominal colostomy is far more satisfactory for the patient and should be recommended.

Surgical treatment for stricture should not be undertaken in the face of active infection. All the acute inflammation must be allowed to subside and the scar tissue should be well established. Treatment in the presence of active infection may lead to exacerbation of the infectious process and more severe complications later on.

In treating incontinence in which the sphincters were divided in the process of operating upon an abscess or fistulous tract the course of action is quite definite, namely, to restore the rectum and sphincteric mechanism as nearly as possible to their former state. The more nearly this can be accomplished the more nearly perfect will be the end result. To explain more readily our conception of the factors of continence I hope we may be pardoned even

by a group of proctologists in briefly recalling the anatomy and function of the terminal portion of the alimentary canal. The normal rectum is a large distensible balloon-like organ lying in the hollow of the sacrum. Its function is to store reasonable amounts of stool or flatus without causing a noticeable defecation reflex. Aiding in this is the fact that the terminal portion of the rectum joins the anal canal at an acute angle often greater than a right angle. The accumulating stool therefore lies in the hollow of the sacrum without pressure on the dentate line until a considerable amount has collected.

The anal canal is a slit-like tube, the sides of which lie in apposition partly from its slit-like construction and partly from a slight recurrent tonic contraction of the sphincteric mechanism. The puborectalis portion of the levators serve a dual role in the maintenance of continence. The two portions of this muscle in contracting cause pressure on the sides of the lower rectum and upper anal canal holding the walls in apposition. It also serves in bringing the cephalad portion of the anal canal upward and forward, increasing the angulation of the point of juncture of the anal canal and rectum. As has been stated, this angulation is important in preventing any appreciable pressure on the dentate line until the rectum is quite filled with gas or stool, producing a definite defecation reflex. Its function is to allow the storage of considerable amounts of stool or flatus without causing a noticeable defecation reflex. No one believes that the muscles surrounding the lower rectum and anal canal are the mechanism which maintains interval continence. No muscle is capable of remaining in a state of constant contraction. There is of course some recurrent tonic contraction due to minor stimulation of gas or stool or other stimuli which is probably a factor in interval continence. In time of need the sphincters may be contracted voluntarily to act as a temporary control against the expulsion of gas or stool; but if pressure is too great or too prolonged, a social catastrophe is the result. This occurs regardless of the frantic efforts of the unfortunate individual to avert such a calamity.

Continence, then, depends upon: (1) The capacity of the sigmoid colon and rectum to store fecal material or flatus; (2) the angulation of the anal canal in relation to the rectum; (3) the anatomic disposition of the musculature surrounding the lower rectum and anal canal; (4) the slight imperceptible recurrent contractures of the sphincteric mechanism and (5) voluntary contractions of the sphincteric mechanism for short periods counteract the effect of a marked defecation reflex. A successful operation for incontinence must restore the rectum to a large, distensible reservoir, reconstruct the anorectal angle and repair the sphincteric mechanism. Unless all three objectives are attained the result will not be entirely satisfactory.

OPERATIVE PROCEDURE

A discussion of the operation for incontinence due to division of the sphincter should be divided into two parts. Those defects which are in the posterior midline will be discussed separately from defects in the other quadrants. The fundamental principle of repair of sphincter defects anteriorly and in the lateral quadrants is primarily to restore continuity and mobility of all the divided muscle. In these cases the capacity and distensibility of the rectum are not likely to have been impaired nor has the posterior anorectal angle been disturbed. In these cases the first step of the operation is to incise the scar tissue and identify the divided ends of the sphincter muscles. The next step is to free the muscle from all adhering scar tissue. It may be necessary to carry the dissection half way around the circumference of the outlet in order thoroughly to free the muscle from scar. In any event when the dissection is complete, mobility of the muscle must not be hindered by attached scar and the operator should be able to approximate the divided muscle without any tension. While the muscle must be well freed from scar, a small amount of scar tissue should be left attached to the severed muscle ends. The reason for this is that the sutures used to approximate the severed muscle will hold more firmly if a scar is left than if they are placed through muscle tissue alone. It should be stated that more failures result from doing too little dissection rather than too much. The choice of suture material is up to the individual. We employ interrupted sutures of No. 00 or No. 000 forty-day chromic catgut. Small gauge wire may be used but occasionally one or more of the sutures must be removed later because they erode through the anoderm and cause a great deal of pain. Chromic catgut sutures

will last until healing is secure and we do not believe that unabsorbable sutures are of any real advantage. The ideal number of sutures to use is the smallest number that will accurately approximate the tissue. Too many sutures or sutures tied too tightly strangulate tissue, producing necrosis and infection which causes destruction of the suture line. If the body of the muscle has been completely freed from the scar, three or four well placed sutures will often be sufficient to approximate the sphincter and allow healing. Any remaining tissue may be approximated on the same principle of overlapping as employed in repairing a ventral hernia. The perirectal tissues can be closed with fine gut or the wound allowed to heal secondarily. We have found infection is less likely and healing is facilitated if the surface wound is left more or less open as has been suggested by Rosser.

In posterior midline defects two conditions are present which do not appear in the lateral and anterior defects. These are damage to the rectal reservoir and damage to the anorectal angle. The extent of these defects depends primarily upon how deep or extensive a posterior incision has been made in the treatment of fistula. With a deep incision the anorectal angle may be completely destroyed and the rectum and anal canal would become a straight perpendicular tube extending from the rectosigmoid juncture to the anal opening.

The basic idea for repair of posterior midline defects which cause incontinence was suggested by observation of patients who had had rectal operations utilizing the posterior midline Harrison-Cripps' incision. These patients with few exceptions had continence equal to that prior to surgery. In the repair of incontinence due to posterior midline defects an attempt is made to duplicate the method of closure following a Harrison-Cripps' incision. To accomplish this an incision is made through the scar caused by the fistula operation to a point well above its cephalad termination, including a short section of the posterior rectal wall. This incision should be completely through the scar tissue. The sphincter muscle and any portion of the rectum which is involved is now entirely freed from scar tissue. Absolute mobility is necessary if the rectum is to be reconstructed as an elastic distensible pouch. All musculature surrounding the lower rectum and anal canal must be mobilized so that traction on the cut margins will cause the muscle to move freely without scar tissue restraint. Again, only a small amount of scar is left on the ends of the muscle to anchor the sutures. Thorough mobility of all tissues is essential to success.

The rectal mucosal defect is now closed with No. 000 chromic suture as far as the dentate line. The suture of the muscle is begun at the cephalad end of the wound and carried caudad to the lower margin of the internal sphincteric ring. It is not always possible nor is it necessary to identify each component muscle which surrounds the lower portion of the rectum. It is necessary, however, to approximate the entire muscle group at their approximate point of division so that the anorectal angle is adequately restored. The adequacy of this angle is determined as the sutures are placed and tied. When completed the cephalad opening should be well anterior and the rectum be posteriorly adequate in size, mobile and resting in the hollow of the sacrum. The margins of the external sphincter are now approximated with a few chromic sutures. The skin margins are not usually closed.

The preoperative preparation of patients with incontinence should be the same as for patients who are to have colon surgery. A low residue diet is given for several days. Sulfasuxidine® (gm. 3) is given four times a day for one week and neomycin® (gm. 1) four times a day is added forty-eight hours preoperatively. Cleansing enemas are given the night before surgery. A saline enema to cleanse the rectum and rectosigmoid is given early on the morning of surgery.

The postoperative care should aim to prevent infection in the wounds and prevent disruption of the repair. Postoperatively the patients are kept on sulfasuxidine (gm. 2) four times a day to keep the bowel as nearly sterile as possible and to maintain a soft stool which can be passed easily. A liquid diet is given for three days postoperatively and then it is followed by a low residue diet. If the patient does not begin to have soft stools and begins to be uncomfortable, warm mineral oil may be instilled through a small urethral catheter. This can be followed by an oil-water enema. The patient is cautioned against straining to expel hard, formed stool. Digital examination and instrumentation is completely avoided until healing is secure. There should not be any

need for instrumentation for two or three weeks after surgery. The stools are kept soft until all the areas are completely healed and there is no danger of wound dehiscence due to constipated stool.

SUMMARY

In summary it is again pointed out that the most common complications of anorectal infections are stricture and/or incontinence. The treatment of rectal stricture is briefly outlined. The treatment of anal incontinence as related to anorectal anatomy is reviewed. A method of handling incontinence due to posterior midline sphincter defects has been outlined.

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DISCUSSION OF SYMPOSIUM ON ANORECTAL SURGERY

R. A. Scarborough (San Francisco, Calif.): In regard to Dr. Hill's paper, I enjoyed his outline of the various etiologic factors in perianal sinuses. I think it is important to all of us, and it is also important to have this available in our teaching on the subject. I was of the opinion, as he concluded his paper, that he left something out; and then, rather apologetically, he presented a case history of a patient which he did not include in his paper.* We have had two similar cases and I would like to assure him that the perianal sinus is going to stay healed since he has resected the segment of the sigmoid.

In regard to the etiology of hemorrhoids, I know I cannot prove my point. I simple want to say that although I follow Dr. Nesselrod 100 per cent in all of his ideas, I cannot see how things he has demonstrated in regard to crypt infection and its complications, can apply in the matter of etiology of hemorrhoids. I know there are a hundred different reasons given for the formation of hemorrhoids in various textbooks. In my experience it does not seem to be correlated with infection. Both infection and varicose veins of the lower rectum and anal canal are very common, but to me hemorrhoids still represent varicose veins due to the fact that we stand in the upright position instead of hanging by all fours from the limbs of trees.

* This case report is included in Dr. Hill's published article.

William J. Rosser (Birmingham, Ala.): I do not know whether it is legal for me to discuss this symposium or not but, Dr. Hill, I believe, I am not the only one here who would like to know the present status of our beloved term "pyoderma." Has that been swallowed up by the hydradenitis suppurativa or is there a separate entity of that type?

Willard H. Bernhoft (Buffalo, N. Y.): I wish merely to add two etiologic factors. When we x-ray a pelvis for osteomyelitis, let us x-ray the lumbar spine. Not infrequently tuberculosis of the spine will point in this particular region and give rise to a situation which has to enter into the differential diagnosis. I have found sinuses, resulting from abscesses draining in this particular region, that lead down to the ischial tuberosities.

Patrick H. Hanley (New Orleans, La.): I, too, would like to add just another etiologic factor in perianal sinuses to the paper of Dr. Hill. I know it is a very rare condition but the possibility of foreign body has to be considered. I have particular reference to a case of ours after surgery in which the foreign body, a sponge, had migrated into the presacral space, necessitating drainage over a period of time until the real cause was discovered by radical exploration of the presacral space.

J. R. Hill (Rochester, Minn.): I should like to answer Dr. Rosser's question. My understanding is that pyoderma includes all skin and subcutaneous infections in that region. At least, that is what my friend the dermatologist says, and hydradenitis suppurativa is only one of those infections.

Rachelle Seletz (Los Angeles, Calif.): I fully appreciate Dr. Nesselrod's magnificent work and I agree with him to a great extent, but I believe we have a tendency to exaggerate this and forget the opposite direction. I still believe in cryptitis. One of the main origins of cryptitis is the deficiency states.

I can always recognize beginning trauma in anal regions by an appearance comparable to inflamed gums. Even dentists have realized now that people under tension and in the

deficiency states are more apt to get so-called trench mouths, and all the other malicious implications they had before have now been cancelled. A man under tension is more apt to get these than one who is kissed and infected by a person.

The same is true of an anal region. I have learned to recognize beginning inflammation of trauma to the anal region in people who are deficient and under tension. When these are corrected, we can prevent a great deal of infection that would radiate into the tissues.

Another thought I have is why not suspect that these special glands may be inclusion glands which do not have a free opening to the surface; and when the secretion is stimulated, these glands will ulcerate and break into the tissue or directly into the crypt.

In regard to the origin of hemorrhoids, I think many of us have forgotten the writing of Dr. Pennington. In his old book he brings it out very strongly and I think Dr. Buie has mentioned it since, namely, we forget the retrograde depression of the vein being compressed as it penetrates the rectal wall on the way to the superior—at least the superior hemorrhoidal vessels on the way to the inferior mesenterics. I see a number of musicians and artists in my practice and I notice that the musicians who are always sitting under tension and watching the baton are the ones who have the largest hemorrhoids.

I hope to bring out one point in regard to prophylaxis of beginning infections. I have used this especially in regard to threatening infection in cases of ulcerative colitis. I find that mucosal application of sulfathiazole ointment has proved valuable to me in many instances of beginning infection or threatening infection especially, as I said before, in cases of ulcerative colitis.

W. C. Wallace (Greenville, S. C.): Dr. Brockman and I believe that the subject of acute cryptitis and the subject of chronic cryptitis are essentially unrelated. We see acute cryptitis following enemas, in cases of dysentery or diarrhea, and we also see it following the marvelous antibiotics we have been using in the last few years. They offer us very little difficulty. We usually clear them up reasonably well in fairly short order, even those from the antibiotics.

I think the subject we are interested in is the question of chronic cryptitis. We agree with Dr. Nesselrod that the crypts, or at least the anal ducts and anal glands, are the source of our abscesses and our fistulas. However, we do not believe that chronic cryptitis develops in a matter of a few weeks or a few months or that it is necessarily a consequence of acute cryptitis. We believe also that the bacteria ultimately present in the bowel are not the bacteria that caused the cryptitis. Perhaps it may not be bacteria at all. Nevertheless, it is our opinion that the chronic cryptitis we see with the thickened valves and the sinus, and perhaps a little secretion coming from it, arises over a long period of years from material that is swallowed and carried through the bowel in sufficient quantities to cause an inflammatory reaction. This creates scarring, weakens the base of the crypt and allows deepening—and the deeper it becomes the more infection is allowed to get into the crypt. Finally, agreeing with Dr. Seletz, a state of malnutrition, avitaminosis, and prolonged strain result, causing this chronic infection to spread into the glands and break through, perhaps block up and give us our acute manifestations.

To conclude and answer the question on hemorrhoids, we all have our own theories. It is my theory that ancestry has more to do with it than any one thing. Most of my patients with hemorrhoids are not constipated until after they have hemorrhoids. We had a paper several years ago on the fact that hemorrhoids were probably due to an excessive arterial blood supply with insufficient veins to carry it back. We inherit poor eyes; certainly heart disease runs in families; why could not hemorrhoidal disease be hereditary?

W. C. Bernstein (St. Paul, Minn.). I should like to have one point cleared up for me and for the other younger members of the Society.

Dr. Thiele, a few moments ago you asked for someone to get up and state what he believed was the cause of hemorrhoidal disease. Dr. Scarborough stated what he thought was the cause of hemorrhoids. Am I and the rest of the group here supposed to assume that the presence of hemorrhoids and hemorrhoidal disease are one and the same thing? I for one cannot accept that thesis. Certainly those of us who are doing routine proctoscopic examinations every day realize that a very large percentage of the population have demonstrable hemorrhoids, and certainly only a small part

of that group have symptoms of hemorrhoidal disease, manifested by pain, bleeding, protrusion, discharge and itching. If we leave this meeting with the idea that everyone who has demonstrable hemorrhoids has hemorrhoidal disease, I think that we are creating a wrong impression.

EDWARD I. LEVY (New York, N. Y.): Dr. Nesselrod, in his brilliant presentation of infection as the cause of hemorrhoidal disease, has opened the question of evaluating the fundamentals in the consideration of low rectal disorders. In such a discussion the normal physiologic trauma to the hemorrhoidal area deserves a place.

The conjoined longitudinal muscle, made up of levator ani fibers and longitudinal fibers of the gut partly attach at the caudal line of the internal hemorrhoidal veins to the rectal submucosa, spraying out to the perianal zone as the familiar corrugator ani. During defecation there is a great spreading of this musculature to allow the oncoming contents of the rectum to pass. The levator ani muscle from its various origins forms a common attachment on the posterior wall of the rectal canal and the sacrococcygeal vertebrae. The posterior rectal wall is subjected to the greatest strain and suffers most frequently from fissure, fistula and hemorrhoids.

A study on intra-abdominal pressure has recently been reported from Germany. It was noted that the intra-abdominal pressure from the dome of the diaphragm to 10 cm. above the umbilicus, the zero level, is negative due to the negative intrathoracic pressure above. Below the 10 cm. level the pressure is above atmospheric pressure. The pelvis is then supporting a pressure that is the product of the distance from the zero level to the pelvic floor times the area of the pelvic floor. Every time the diaphragm contracts, the pressure on the pelvic floor and its organs is increased. The venous return of the rectal wall is by veins which have penetrated the wall about half way down the rectum, and then proceeded within the muscularis until they achieve the inner wall as the veins of the rectal columns, the internal hemorrhoidal veins. These veins do not have valves. Increase of intra-abdominal pressure by diaphragmatic descent pushes a long column of blood against this wall, distending the veins. Upon release of the obstructing pressure, the wall is restored to its previous state by its elastic recoil. If elasticity is lost, the veins form the bulbous enlargements characteristic of the internal hemorrhoidal condition.

Therefore, consideration of the pathogenesis of low rectal disease, exclusive of neoplasia, should include the factor of physiologic trauma from (1) the spreading of the conjoined longitudinal muscle in defecation and (2) the rhythmic rectal venous obstruction caused by the contraction of the diaphragm.

ELMER E. WADSWORTH (Alhambra, Calif.): I should like to pose a question. One of the most common sites of the deep crypt that I find in a patient is on the left side, nevertheless those other etiologic factors are not present to produce a fissure there. Why don't we get fistulas arising from that area and the other things that would arise from the inflammatory processes that stem from cryptitis? I am sure that the problem has posed itself to him but I just would like an answer if he has it in that particular phase.

A. G. SCHUTTE (Milwaukee): I would like to ask Dr. Nesselrod on the indirect spread of the bacteria along the lymphatics and the blood vessels, if that couldn't account for a fistula without an internal opening?

J. P. NESSELROD (Evanston, Ill.): I had sincerely hoped that this thesis on anal infection would create some argument and I am very pleased that it has. May I remind all of the discussants that I purposely left out a number of contributory factors, not because I think they are unimportant or uninteresting but because there just wasn't time to go over all those things. I heartily agree with Dr. Scarborough and Dr. Seletz that venous pressure has a great deal to do as a contributory cause of hemorrhoidal disease.

Quite a number of my medical confreres have said to me, "Nesselrod, why isn't it possible for a patient who has a cirrhotic condition of the liver or some other form of portal obstruction to have his hemorrhoidal disease on the basis of such a factor"? In answer to this question, let me ask another. The superior hemorrhoidal veins are tributary to the portal system, whereas the middle and inferior hemorrhoidal veins are tributary to the caval system. Therefore, would it not seem that in the presence of obstruction to the portal system there might be a means of escape of blood via the middle and inferior hemorrhoidal veins? A

common cause of obstruction in all three tributaries is pregnancy toward term in which we see a good deal of hemorrhoidal disease in women.

DR. WADSWORTH: I realize that I am just a boy in experience compared to Dr. Rosser, but I do think that he is giving us a rule which, like all rules, should be broken at times. Primary fistulectomies or fistulotomies should be done on a selective basis for each individual case. I know a number of times I have seen it when it seemed quite justified, and all the other factors being included we got a very good result and saved time, money and tissue.

In my experience over the last six to eight years as consultant and operating surgeon at Olive View Sanitarium in Los Angeles, I have had an opportunity to see a great deal of tuberculosis of the anus and rectum, but now we are having a markedly decreasing clinic out there. I find also that the nose and throat clinic is smaller. Because of their triple hemotherapy they are doing chest surgery on fewer people than before. I have seen ulcerative colitis of a tuberculous nature on one visit and a month later could not believe it was the same bowel. That condition is practically cleaned up.

I want to emphasize that I have performed a great deal of surgery on these tuberculous anorectal lesions. One of the bases for doing so is that by proper drainage and cleaning up these lesions I relieved the body as a whole of that extra effort; and I know of specific instances in which the patients started to do better immediately as far as their chests were concerned after rectal surgery.

HOWARD K. BELNAP (Ogden, Utah): I should like to ask Dr. Rosser if he has found any practical means whereby we can run sensitivity tests in the office? At home I send my patients to the laboratory for these tests and it costs them $15.00. At the same time it takes four to five days to get a report. If there is a means of performing these tests as an office procedure with the use of an incubator of some type, it would be very practical. If Dr. Rosser has that, I would like to know.

I agree with Dr. Wadsworth that there are many cases of acute abscesses in which an opening of the crypt is justified when draining the abscess. Undoubtedly you have all seen some cases in which at the point of operation you find a little bit of pus escaping from a crypt in the immediate vicinity of the abscess. Opening and draining that crypt and starting that as your drainage point for the opening and draining of the abscess results in a final cure of the disease.

DR. ROSSER: I am sure the bell saved me from some more comments concerning the question of primary fistulotomies in abscesses, because I do know that this is a thing which has had some powerful backing in the United States.

I call attention to the fact that both Dr. Wadsworth and Dr. Belnap say that in certain definite circumstances they believe this might be the proper thing to do. I would not go so far as to say that it is not feasible; but the type of literature coming out lately suggests its use in all circumstances, and that is the particular thing to which I wanted to call your attention.

FRANCIS S. ADAMS (Pueblo, Colo.): Mr. Chairman, I am very much stimulated by all the papers but particularly by the last paper. First, there is the question of semantics. Many patients who come into your office complaining of loss of bowel control do not mean that at all. Sometimes they mean simple powerful urgency toward defecation and treatment directed toward incontinence, of course, would be misdirected. Probably the patient has proctitis or something similar and has urgency with no loss of control whatever.

In true cases of incontinence I am impressed by the importance of the contraction of radially directed scar tissue. Contractures which proceed radially to the anus are deforming, and often these contractures or scar tissue are attached at their distal end to nearby bone, as the ischial tuberosity. Relief of this contracture is essential for the restoration of the normal contour of the anus. Many of these cases have the anus perfectly round except in the direction of the contracture where there is a notch in that direction. Often a big block of scar tissue has to be removed.

In most of these cases I believe I have satisfactorily traced the cause of this contraction to suppuration which has been allowed to continue too long in previous treatment or too much skin has been removed in the treatment of either the abscess or the fistula. The only course that nature has left in healing is to let down a great contracting block of scar tissue.

I do not believe at the present time, except in some fairly large centers, that there is a

good deal of interest shown in this laboratory procedure. We are not at this time in a position to get a sensitivity test quickly. In one of our hospitals in Dallas they are quite interested in sensitivity tests and I believe if this procedure is more generally used by clinicians that laboratories will follow along and will make their services more available. Of course, you don't want to wait five or six days and you don't want your patient to pay such a large fee; but the test is becoming much simpler and I am hopeful that it will be possible to use it in all communities because it should be very helpful.

Infections and Sinuses Other Than Fistulas in the Perianal Region

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THE term "perianal region," used in the title, cannot be exactly defined. As employed here, it applies to a region sufficiently close to the anus that abscesses and sinuses found in the neighborhood could have anorectal connections whether or not such connections actually exist. It includes, therefore, not only the tissue immediately surrounding the anus, but also the adjacent tissue.

Two other concepts, moreover, underlie what is said here: the structure here called a "fistula" is presumed to connect with the anal or rectal lumen; what is here called a "sinus" may be the external end of a fistula or may be a tract that has no connection with the lumen of the anus or rectum. It is with the latter type of sinuses that this paper is primarily concerned.

When the proctologist sees a patient with an abscess or a draining sinus in the perianal region, the burden is on him to prove that a fistula is not forming or does not already exist. I found that 14 per cent of patients whose perianal sinuses were operated upon by members of the Section of Proctology at the Mayo Clinic were of non-fistulous origin—under the definitions given. The ensuing discussion of non-fistulous sinuses will not be confined to what was learned from the study just mentioned but will be drawn from an experience of conditions encountered, at one time or another, by my colleagues and myself.

INFECTIONS ARISING IN SEBACEOUS GLANDS

Comedo. In this condition sebum plugs the mouth of a sebaceous duct. In the skin about the anus comedones may become relatively large, may undergo inflammation and may give rise to pustules. Occasionally a tiny, superficial sinus remains after this infection. The presence of other comedones usually makes the diagnosis obvious.

Sebaceous Cyst. A sebaceous cyst is attributable to an accumulation of sebaceous matter in the gland itself; in other words, it is a retention cyst. It may undergo inflammation and suppuration and may form a chronic nonfistulous sinus which drains periodically. After a sinus has formed it may not be possible to identify the preceding condition as an infected sebaceous cyst. The history of a painless subcutaneous mass (the size of a pea or larger) having been present in the same location as the sinus, but prior to the onset of inflammation, is suggestive evidence.

Furuncle and Furunculosis. A furuncle is the result of introduction of pyogenic organisms into a sebaceous gland or hair follicle. It is characterized by acute, circumscribed inflammation which usually ends in suppuration and formation of a central necrotic mass, or "core." A sinus rarely, if ever, follows but tender, localized, subcutaneous induration may persist for weeks if the surrounding tissues have been traumatized by the patient's squeezing them to dislodge pus.

The term "furunculosis" refers to the condition that accompanies a crop of boils, a disorder that may extend over weeks or months. When furuncles keep recurring, the history is most suggestive of fistula. Since furuncles, however, do not tend to recur in exactly the same area, and do not leave sinuses, distinction between the two conditions usually is not difficult.

INFECTIONS ARISING IN SWEAT GLANDS

Hidradenitis Suppurativa. In the perianal region, as well as in the axillary, mammary, inguinal and genital regions, some of the sweat glands are larger and more deeply situated in the subcutaneous tissue than is usual and are called "apocrine" glands. Hidradenitis suppurativa is the result of infection of these large

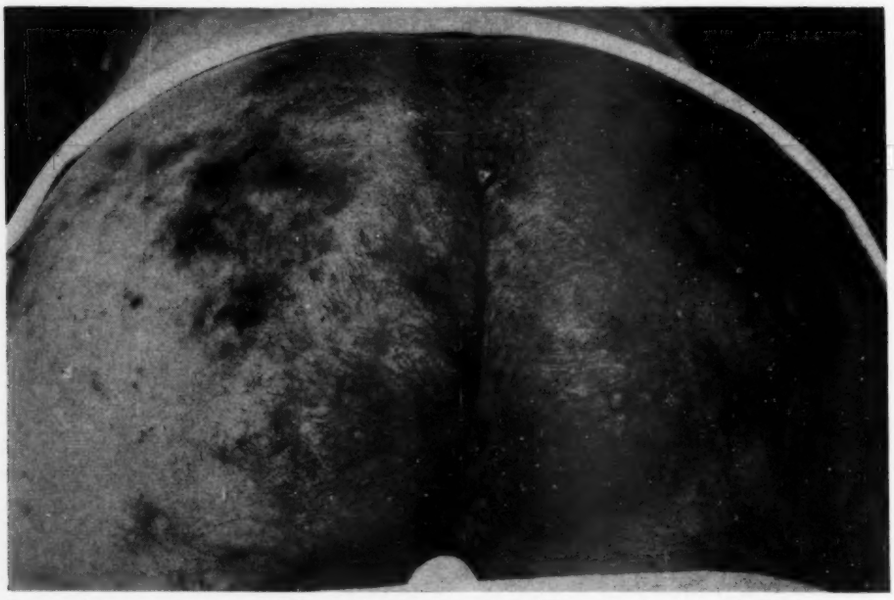

FIG. 1. Advanced stage of hidradenitis suppurativa. (From Jackman and McQuarrie.[2])

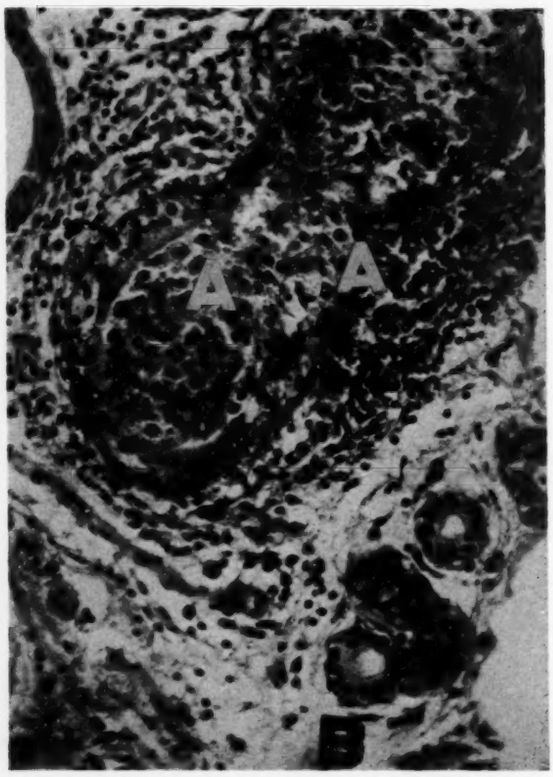

FIG. 2. Early stage of hidradenitis suppurativa showing (A) acute inflammatory reaction within the lumen of the apocrine gland and in the periglandular connective tissue, and (B) absence of involvement of the neighboring eccrine glands. Hematoxylin and eosin, × 240. (From Brunsting, L. A. Hidradenitis suppurativa; abscess of the apocrine sweat glands. *Arch. Dermat. & Syph.*, 39: 108–119, 1939.)

glands. It is manifested in the early stages by one or more subcutaneous nodules, which gradually grow to the size of a pea or larger and undergo softening and suppuration; subsequently, non-fistulous sinuses form. The process spreads through the skin and subcutaneous tissue. In the later stages, it is associated with cutaneous thickening and induration, as well as with honeycombing of the skin and subcutaneous tissue by draining sinuses and abscess cavities. (Fig. 1.) It usually is accompanied by extensive scarring. New lesions, also, frequently may be seen cropping up in the immediate vicinity. The disease may remain localized but, not uncommonly, in conjunction with the perianal involvement, there are lesions in the sacrococcygeal, gluteal, genital or inguinal regions, and in advanced cases there may be mass involvement of all these tissues concomitantly.

Hidradenitis suppurativa is a disease of adulthood, since the apocrine glands do not become active until puberty. In the early phases, the disease is not easily distinguished from other localized suppurative conditions. Then positive diagnosis can be made only by histologic examination, which gives evidence of primary inflammation of the gland. (Fig. 2.) In the chronic stage, the disease, particularly if it is extensive, usually is not difficult to diagnose. It may be confused, however, with extensive pilonidal disease or anal fistula.

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FIG. 3. Pilonidal disease with draining non-fistulous sinus (not connecting with the anal or rectal lumen) lateral to anus. Another such sinus may be seen (arrow) in midline in lower sacral region. The two sinuses were connected by a fibrous, subcutaneous channel.

FIG. 4. Similar to condition represented in Figure 3 except that one non-fistulous sinus is anterolateral to anus. Metal probe is in sinus over sacrococcygeal region.

More often than not the diagnosis of hidradenitis suppurativa will be aided by finding similar lesions in one axilla or in both.

PILONIDAL DISEASE

An abscessed pilonidal cyst ordinarily points over the sacrococcygeal region. Occasionally, however, the infection burrows downward and points in the perianal region, usually posterior or lateral to the anus (Fig. 3) and on rare occasions anterior or anterolateral to it. (Fig. 4.) Such an abscess may be hard to distinguish from one arising within the anus or rectum unless, when the abscess is opened, it is found to contain hair. On the other hand, after the abscess has been opened and sufficient time has elapsed, by palpation or probing or both a fibrous tract readily can be traced to a non-fistulous sinus in the midline over the sacrococcygeal area. This sinus often is tiny. Rarely, there may be coexisting pilonidal disease and anal fistula.

CYSTS ANTERIOR TO THE SACRUM OR COCCYX

Dermoid cysts which are situated between the posterior wall of the rectum and the anterior surface of the sacrum frequently are

FIG. 5. Non-fistulous sinus posterior to anus in patient who had presacral dermoid cyst. A sinus in this same location may be associated with mucous cyst.

accompanied by a congenital sinus (or dimple) just outside the external anal margin posteriorly, in the midline. (Fig. 5.) I have seen only one patient with an uninfected dermoid cyst and, in that instance, there was no drainage from the non-fistulous sinus, one reason being that the sinus was not patent all the way to the cyst. (Fig. 6.) On the other hand, since most dermoid cysts in this region have undergone infection by the time the patient seeks the surgeon, it seems reasonable to believe that a congenital sinus may have served as the portal of entry for pyogenic organisms. Infected

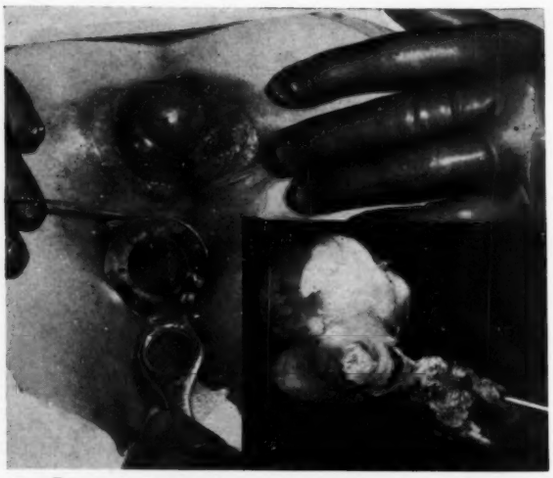

FIG. 6. Uninfected dermoid cyst has been dissected from presacral region and is presenting in wound. Incision extends from posterior perianal region to a point lateral to the coccyx. The latter did not have to be removed. A probe is in the funnel-like non-fistulous sinus and anoscope is in place. Inset: Cyst after removal, with probe in sinus. There are numerous small mucus-secreting cysts adherent to the outer wall of the sinus. (From Hawkins and Jackman.[1])

dermoid cysts may drain into the rectum by rupturing the rectal wall, in which case they should not be included in this discussion, or they may form one or more non-fistulous sinuses in the perianal region, usually posterior to the anus. When the latter takes place, the condition may readily be confused with anal fistula. The presence of a mass between the rectum and the sacrum, and absence of a demonstrable opening in an anal crypt, point to the diagnosis of infected dermoid cyst. The true nature of the condition, however, may not be realized until surgical exploration. Frequently the situation will have been further confused by one or more previous operations for what was thought to be a fistula.

Another type of cyst occurs more frequently than does a dermoid. It is small, usually not more than 1.5 to 2 cm. in diameter; it may be multilocular; it has a thin wall which is lined with columnar or cuboidal epithelium, and it contains a clear, mucinous material. Usually it is situated lower than is a dermoid cyst; in other words, it is found between the posterior wall of the rectum and the anterior surface of the coccyx. This type of cyst probably arises from a remnant of the tailgut and it may be accompanied by a congenital non-fistulous sinus situated posteriorly in the midline, just outside the anal margin. There is no discernible drainage from the sinus and, in my experience, this type of cyst rarely becomes infected.

TRAUMA

There are several ways in which trauma may result in the formation, in the perianal region, of an abscess, a non-fistulous sinus or both. For instance, patients with pruritus may start a furunculoid type of lesion by scratching. Occasionally traumatic fat necrosis also is seen. This is caused by an injury, such as a blow, to the perianal region; the fat cells of the subcutaneous tissue are traumatized, resulting in necrosis, inflammation and finally chronic induration. Such tissue may have to be excised, or at least unroofed, before the process will subside.

Infection leading to formation of a chronic, draining, non-fistulous sinus may be caused, moreover, by some type of foreign body being introduced through the skin into the underlying tissues, particularly if the foreign body is not removed or is not absorbed. The foreign substance may be introduced accidentally, in which case the sinus may be extensive. Or the traumatizing material may be deposited by injection, for treatment of some condition such as anal pruritus, or to produce lasting analgesia after operation on the anus.

OSTEOMYELITIS

Infection arising in the bones of the pelvis may point in close proximity to the anus and result in a draining, non-fistulous sinus. Whenever a chronic perianal sinus is deep, and the cause of it is not readily suggested, a roentgenogram of the pelvis should be made to rule out osteomyelitis.

ABSCESSES AND SINUSES PECULIAR TO THE PERINEAL REGION

In the female, abscesses and occasionally draining non-fistulous sinuses may result from infection of Bartholin's glands. These glands are on either side of the posterior commissure of the vaginal entrance and infections of them usually are readily recognized. However, anal fistulas may be found in these parts and a diagnosis of Bartholin's abscess should not be made until the possibility of an anorectal connection has been eliminated by careful examination. Other causes of perineal non-fistulous

sinuses in the female are faulty healing of lacerations and of episiotomy wounds following childbirth.

In males, abscesses and sinuses rarely may result from periurethral infection and usually are associated with either a benign urethral stricture or a urethral malignant growth. Commonly, a history of urethral instrumentation can be obtained in such instances. After a sinus has formed, the diagnosis usually is simplified by drainage of urine through it.

CASE REPORT

I should like to describe briefly an unusual case which at first was thought to be a non-fistulous sinus. The condition of this patient does not agree strictly with the definitions laid down earlier in this paper, but I believe it is sufficiently interesting to warrant mention and to be recorded.

A sixty year old man came to me because of a chronic draining sinus situated 4 cm. from the anus in the left posterior position. This sinus had been present for approximately a year. It had developed after incision and drainage of what the patient's home physician had called a left "ischiorectal" abscess. Prior to drainage of the abscess there were no symptoms other than pain and swelling to the left of the anus. The patient said these symptoms had been present for a considerable time—apparently several weeks—although he was not able to remember definitely when the difficulty had started. Diagnostic studies included a roentgenogram of the pelvis, which disclosed nothing significant, and roentgenologic examination of the colon, which demonstrated only diverticulosis of the sigmoid. On two occasions I examined the terminal portion of the colon for a distance of 24 cm., once while the patient was under the influence of caudosacral anesthesia. I found nothing significant except that I could trace the sinus tract, by palpation and probing, to a point anterior to the rectum and just above the prostate gland. The question as to the cause of the sinus finally was solved by the injection of a contrast medium (diodrast) into the perianal sinus while the patient was under the fluoroscope. The contrast medium described a connection with the mid-sigmoid, showing that the patient therefore had a sigmoidoperianal fistula. (Fig. 7.) Resection of a portion of sigmoid was performed with end-to-end anastomosis and obliteration of the fistulous opening into the sigmoid. This operation showed that peridiverticulitis was present and that the fistula had been caused by a ruptured diverticulum.

FIG. 7. Oblique view showing catheter in portion of perianal sinus; contrast medium occupies the rest of the sinus.

It is still too early to know whether or not the terminal portion of the fistula will close spontaneously.

COMMENTS

Cause. The cause of most non-fistulous sinuses in the perianal region is evident in the history, physical examination, pathologic findings, or all three. Sometimes, however, the exact nature of the condition is not evident on microscopic examination because the infection has destroyed the tissues involved. The only possible microscopic diagnosis may be simply "inflammatory tissue." Bacteriologic studies usually are unnecessary but they may be the only way to determine the cause of some conditions, such as actinomycosis and blastomycosis. On occasion, also, it may be necessary to call in the radiologist to study extensive sinuses after injection into them of radiopaque substances. The cause of some sinuses remains ultimately obscure.

Treatment. Detailed discussion of treatment is impossible here. In general, however,

antibiotics are effective only in treatment of furuncular lesions and for prevention of the spread of certain other conditions. Once suppuration has occurred more radical measures usually are required and they are likely to be curative even if the cause cannot be ascertained. Acute abscesses should be incised and drained, regardless of cause; if they are chronic, the source of the infection as well as the infected tissue must be eradicated. In some instances merely unroofing a chronic abscess or non-fistulous sinus may suffice but one usually can be more nearly certain of the outcome if the entire process is excised. In cases of hidradenitis suppurativa this may result in an extensive denuded area, which may require subsequent skin grafting. All patients with hidradenitis suppurativa should be told that the disease may extend and that the new lesions should be removed as they occur.

In spite of what has just been said in favor of surgical treatment, the exception must be kept in mind. Occasionally an extensive non-fistulous sinus leads from the perianal region deep into the pelvis but causes very little disability. In such instances, considering the mutilating procedure which would be required to eradicate the sinus, it may be wise to forego surgical treatment and to continue observation.

SUMMARY

In this paper, the term "fistula" is used only if the tract connects with the lumen of the anus or rectum. Under this definition, only 14 per cent of a series of perianal sinuses studied at the Mayo Clinic were of non-fistulous origin. Non-fistulous sinuses generally arise subsequent to, or as accompaniments of, the following conditions: infections arising in sebaceous or sweat glands, pilonidal disease, cysts anterior to the sacrum or coccyx, trauma, osteomyelitis and abscesses and sinuses peculiar to the perineal region. Treatment usually is surgical. However, if a non-fistulous sinus is causing little disability but is so extensive that its surgical eradication would be mutilating, surgical operation should be withheld and the patient kept under observation.

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*Sturnick, M. I., and Gargill, S. L.: New England J. Med. *247*:829 (Nov. 27) 1952.

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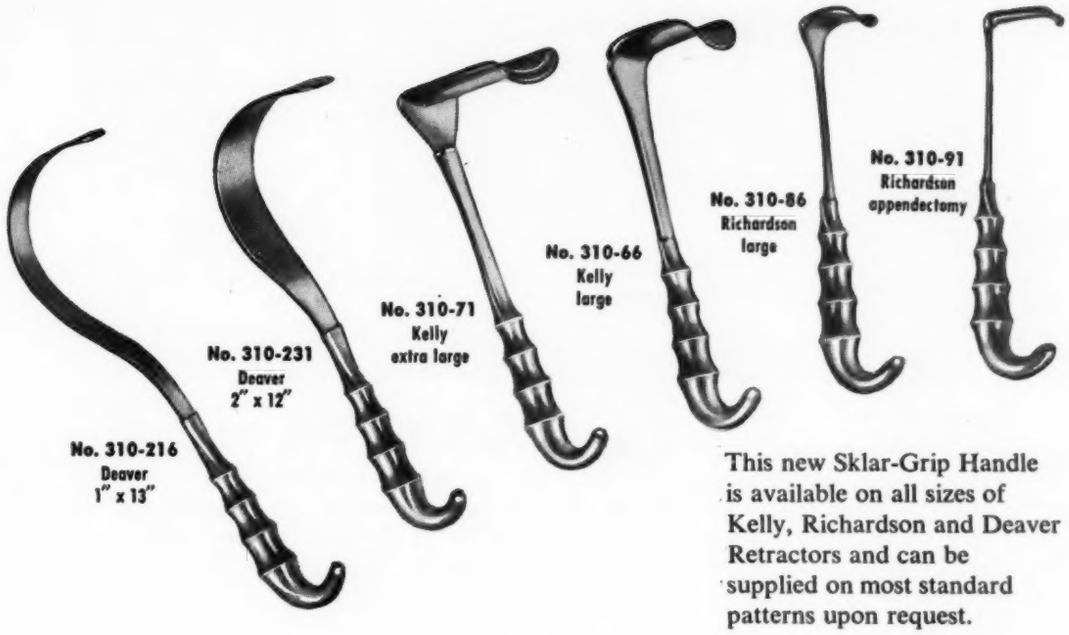

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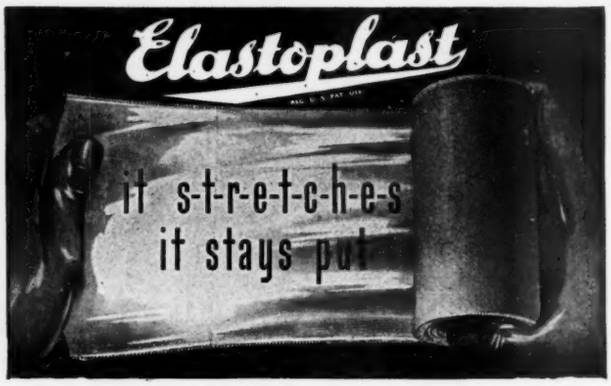

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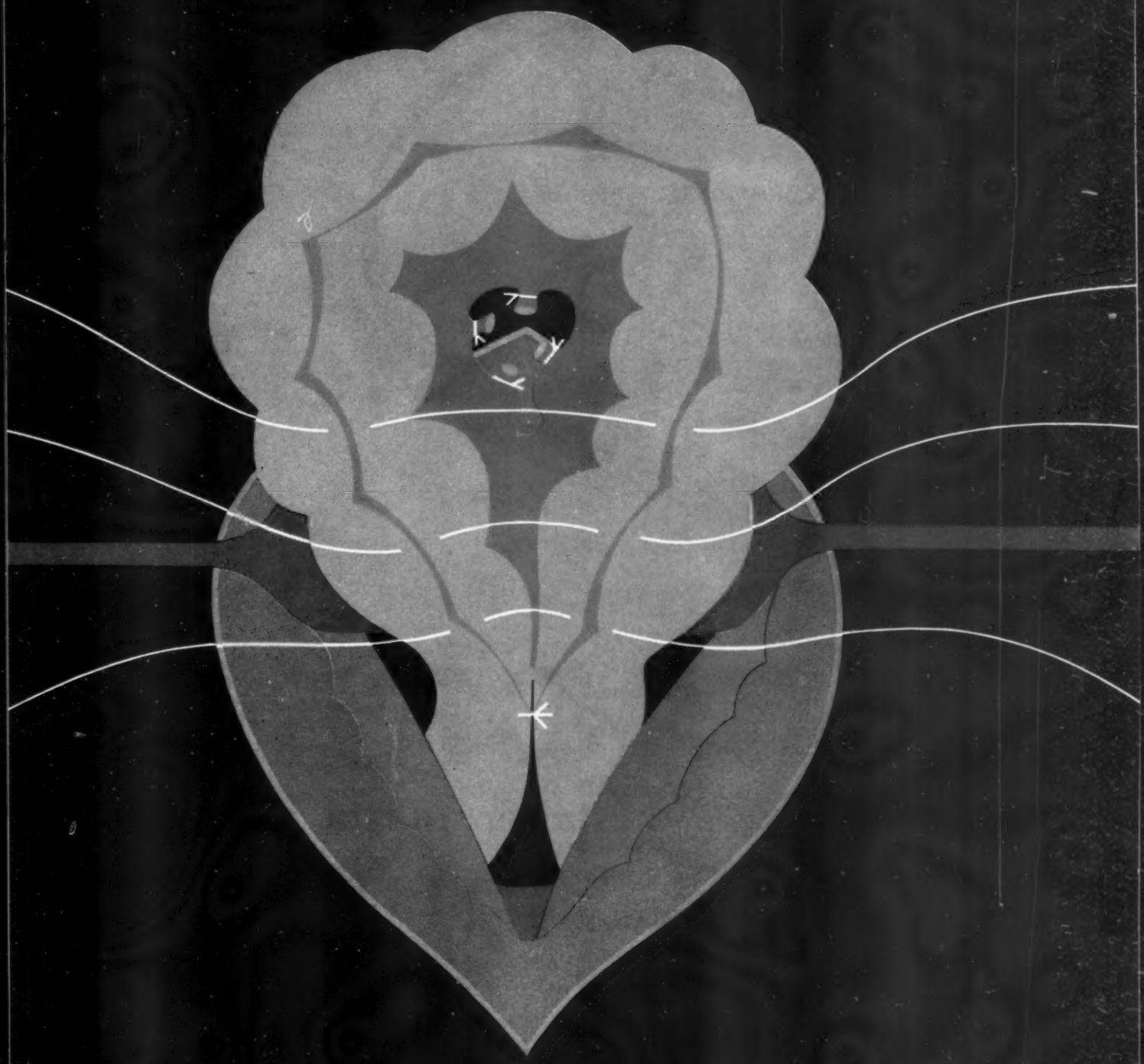

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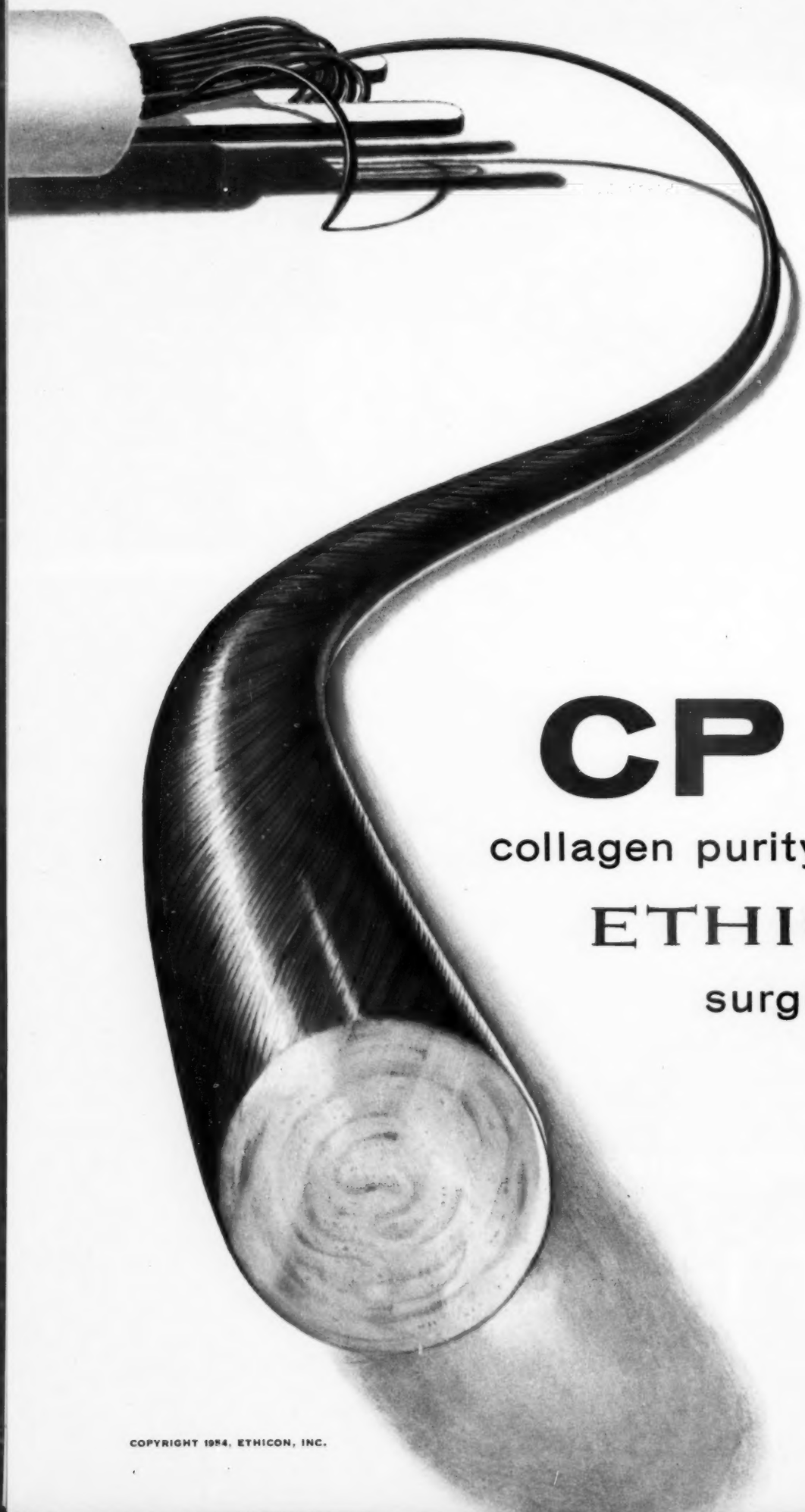

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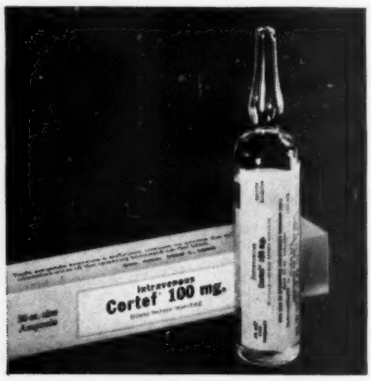

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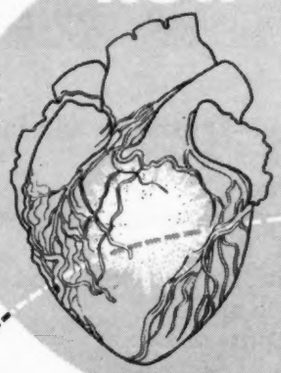

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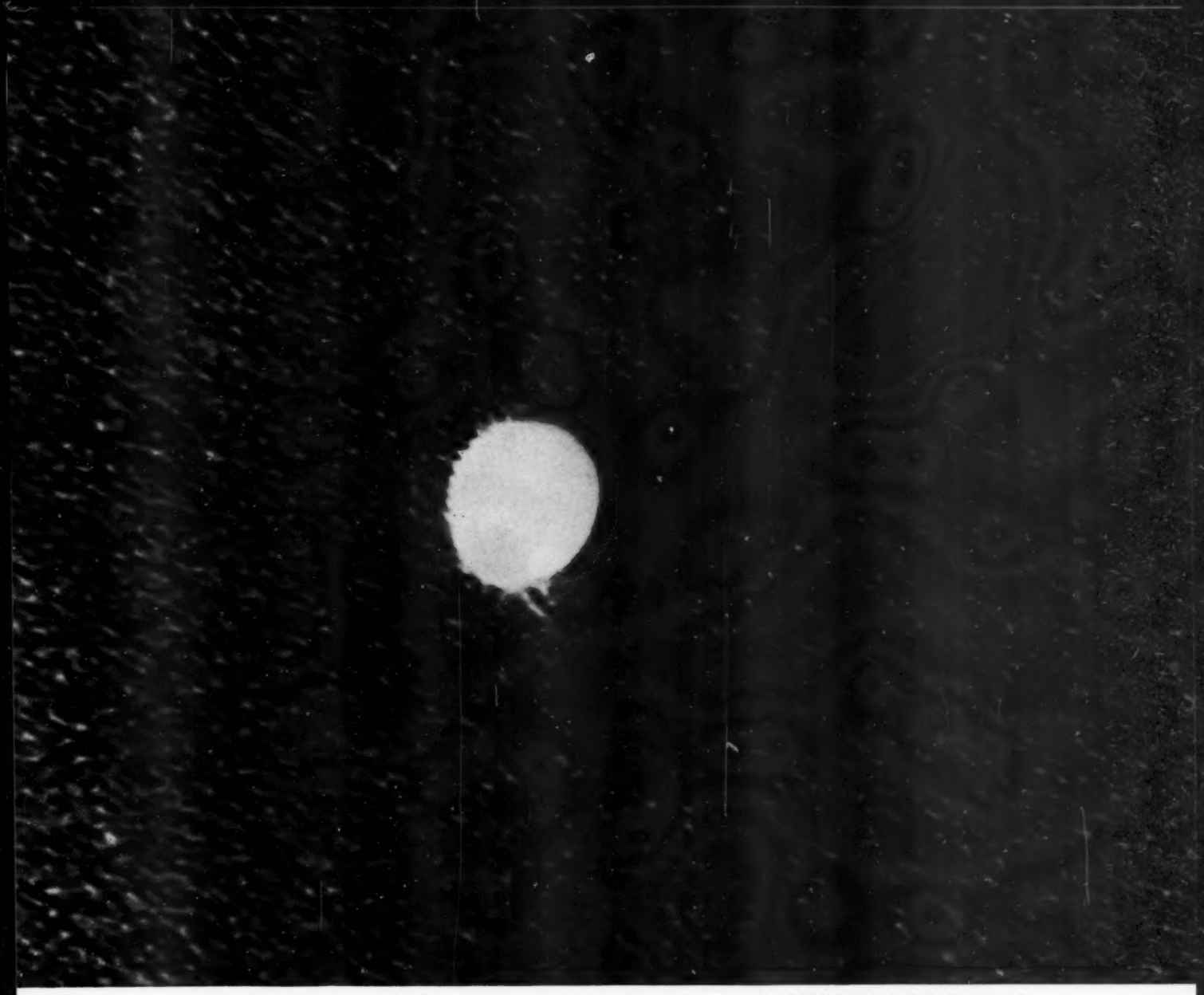

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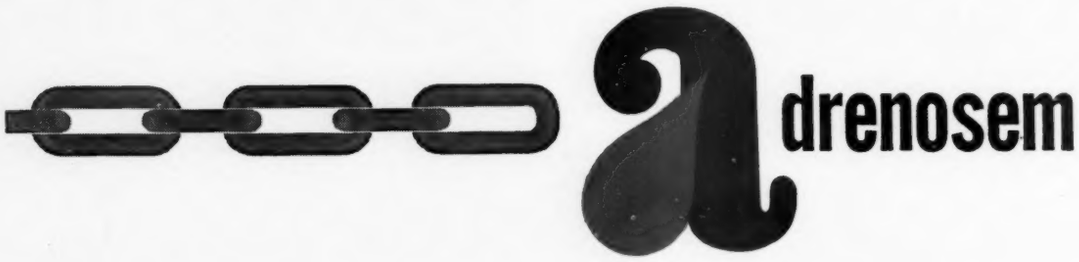

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Diplococcus pneumoniae 35,000 X

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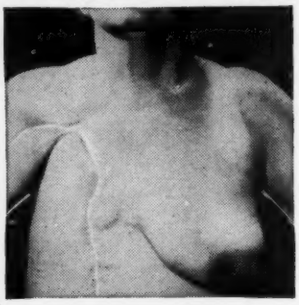

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A candidate for a Proctologic residency approved by the Board must be a graduate of a medical school approved by the Council on Medical Education and Hospitals, and must have served an internship of at least one year in a hospital acceptable to that Council. In addition, he must have had specific training in general surgery after internship, including instruction in the basic sciences according to standards prescribed by the Board.

An applicant for examination leading to certification by the American Board of Proctology may qualify by any one of three methods: (1) two years of approved general surgical residency and two years of approved residency in proctology (colonic and anorectal surgery); (2) three years of approved general surgical residency, plus two years of an approved precepteeship in proctology (colonic and anorectal surgery); (3) two years of approved residency in general surgery, plus three years of approved precepteeship in proctology (colonic and anorectal surgery).

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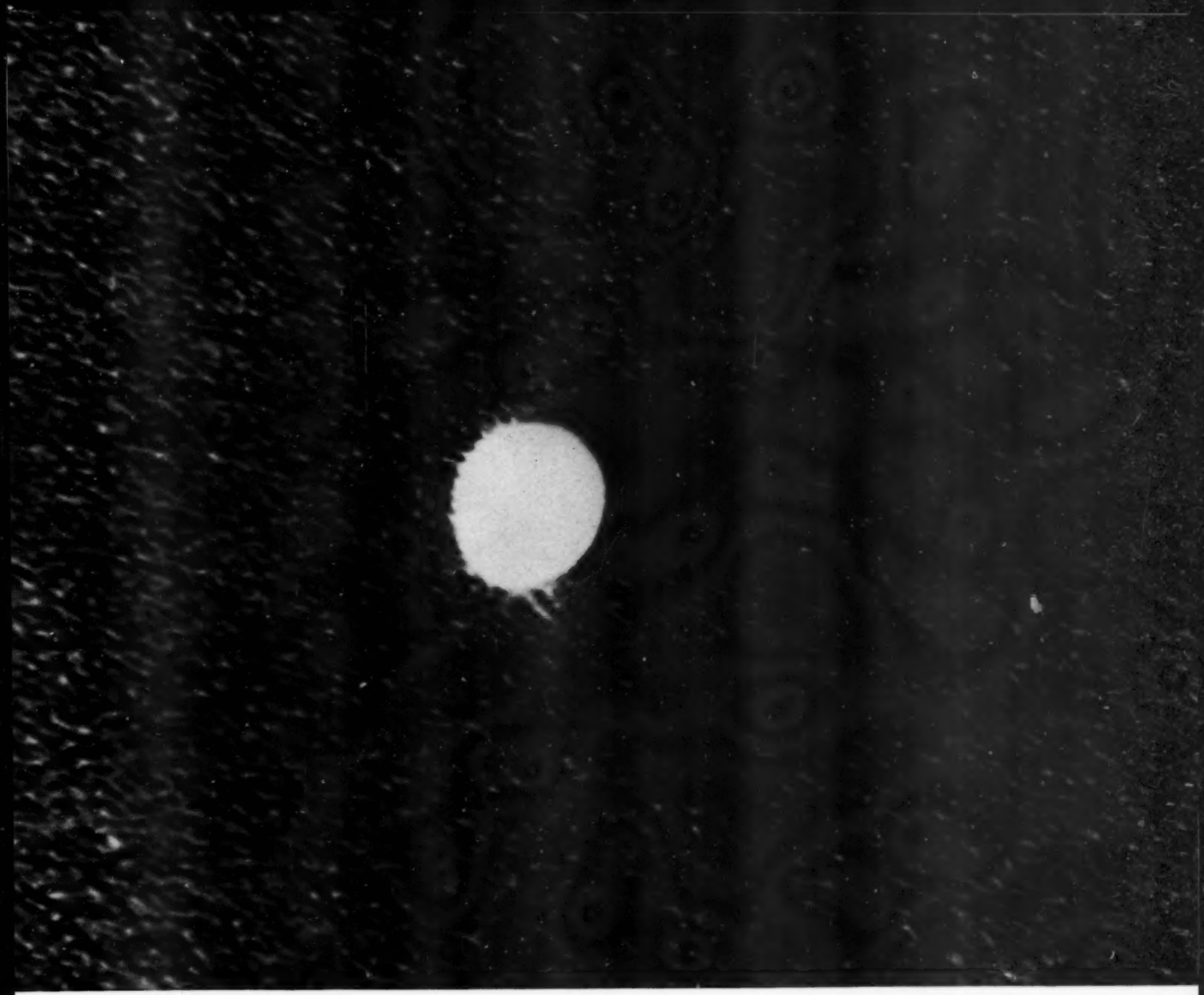

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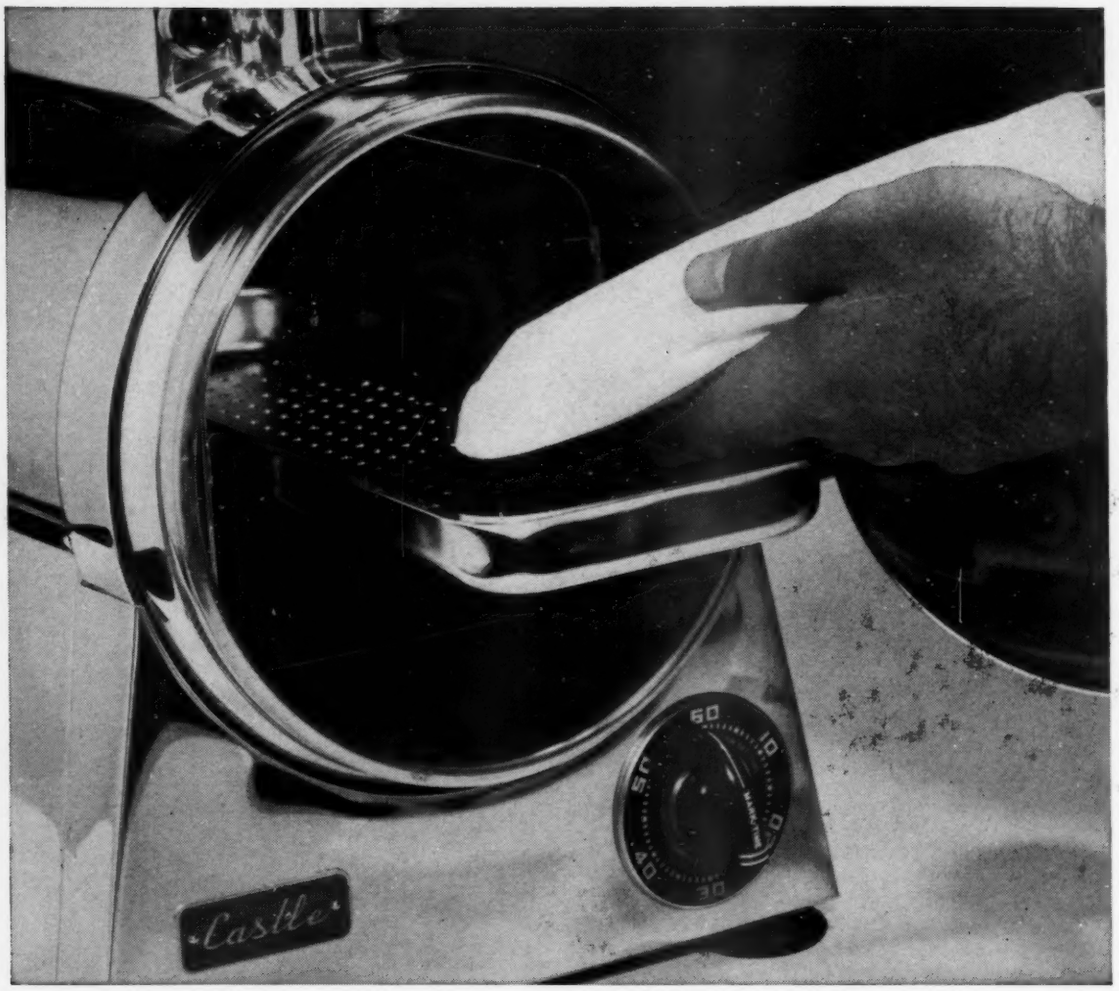

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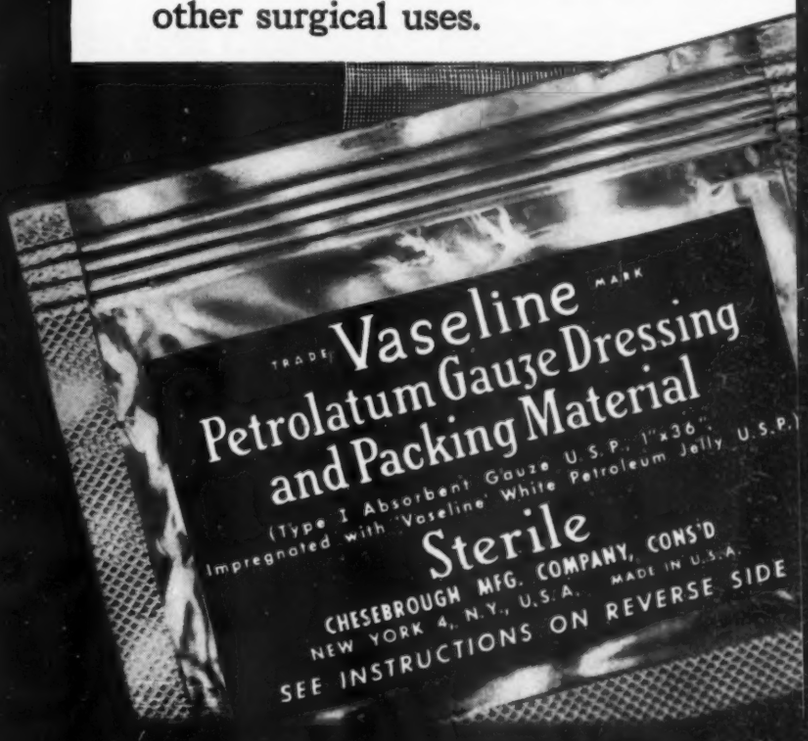
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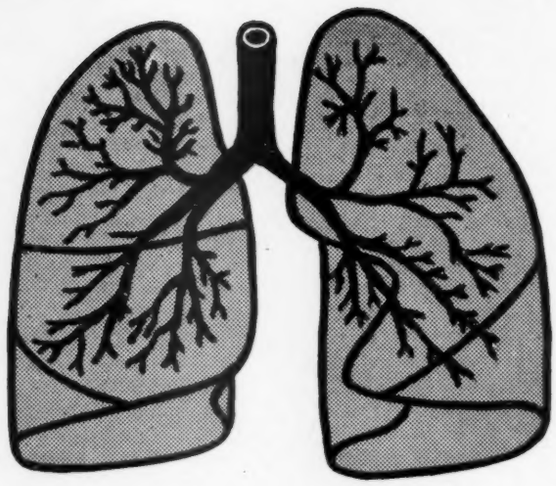

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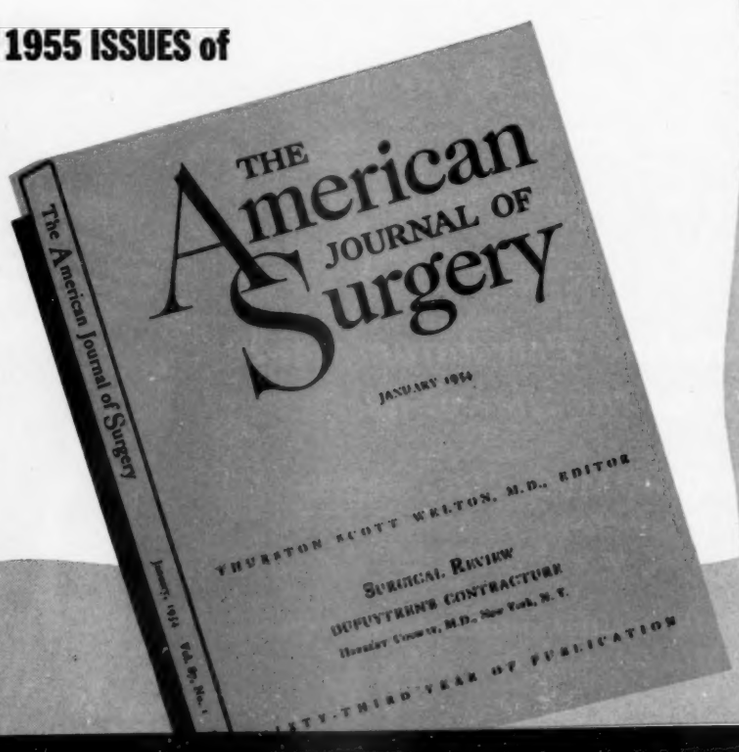

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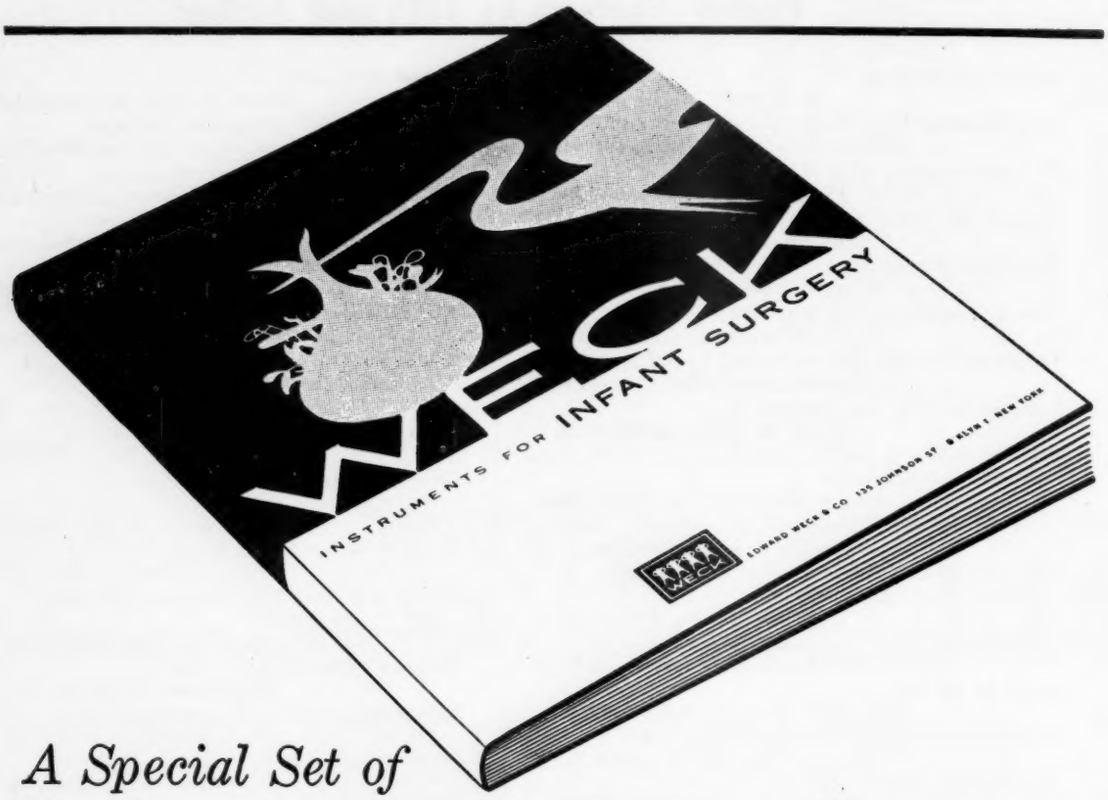

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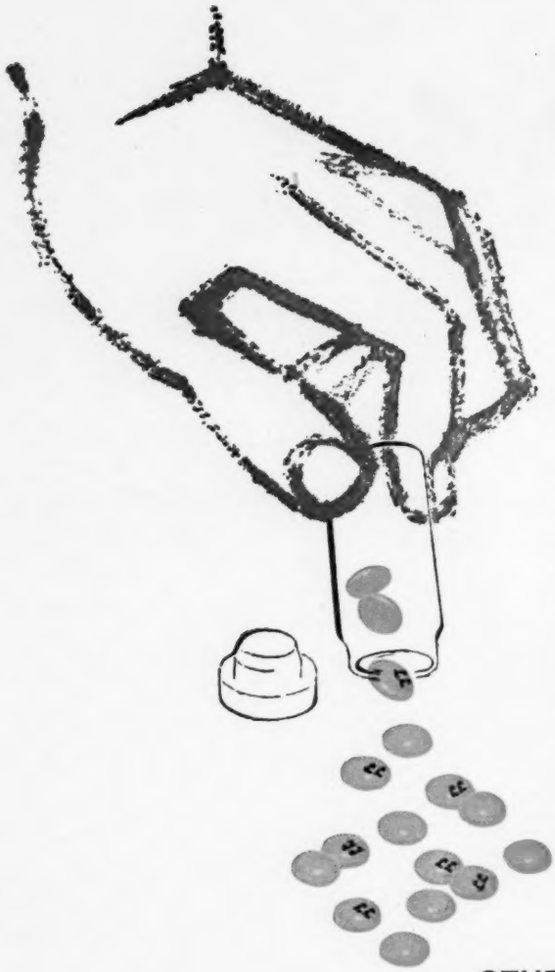

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NOTES AND QUOTES

A tendency to bleed too easily, whether manifested by nosebleeds, bleeding gums or skin that bruises readily, often means a Vitamin C deficiency. Easy bleeding was common among 2,000 women who had lost one or more babies, reports Dr. Carl T. Javert of Cornell University Medical College. One role of Vitamin C is to provide the binding material that reinforces the walls of the small blood vessels or capillaries. "A distinct correlation was found between Vitamin C deficiency and decidual hemorrhage," says Dr. Javert. In this condition there is bleeding of the vessels in the lining of the uterus during pregnancy. Treatment, as outlined by Dr. Javert, includes a diet rich in citrus fruits.

MILLIONS of Americans participate in competitive athletics either as individuals or as members of a team. Judging by available information on the numbers who take part in different sports, the most popular are baseball, basketball, golf, tennis, football, boxing, and track and field events. Large numbers of enthusiasts also take part in skiing, soccer, wrestling, hockey, rowing, lacrosse and fencing. Some of the participants in the various sports are professionals—that is, they earn their livelihood thereby—but the vast majority engage in the games solely for recreational purposes.

With so large a participation in athletics it is not surprising that sizable numbers of people each year are more or less seriously injured in such activities. While no over-all estimate of the injured is available, it is clear from scattered sources that the national toll of those who suffer fractures, sprains, strains, cuts and concussions mounts to tens of thousands annually. Fortunately, however, fatal injuries are relatively few. For example, among the several million males between the ages of ten and twenty-nine insured under Industrial policies in the Metropolitan Life Insurance Company, there were only eleven deaths resulting from competitive athletics in the two years 1951 to 1952, or less than one in every 300 accidental fatalities of all types in this insurance experience. Baseball and softball accounted for four of the deaths in sports; golf, three; basketball, two; and football and track, one death each.

The establishment of "Lakeside Academic Prizes" for the Marquette University Medical School including an unusual commendation for a laboratory technician, has been announced by Harvey L. Daiell, M.D., scientific director of the pharmaceutical firm. Three prizes will be given each year and will be awarded non-competitively, on the basis of academic achievement alone. The three include $100 to a lower classman, $100 to an upper classman and a $50 prize to a laboratory technician. The latter award is believed to be the only one of its kind.

"We hope that the prize to the laboratory technician will encourage further recognition of his important role," Dr. Daiell explained. "Through research, the laboratory technician helps the medical school to train better physicians and to make contributions to medical science."

AN advertisement sponsored by Parke, Davis & Company seeks to help small communities needing a doctor and young doctors looking for a place to settle. The advertisement is headlined, "Does Your Community Need A Doctor? If so, the physician you're looking for may be the one who's looking for you!"

The copy points out, "Somewhere there is a doctor who would like to live in your community. He may be completing his internship and looking for a place to settle. He may be a resident physician in a big-city hospital, ready for a private practice. Or he may be a metropolitan specialist who longs for a chance to get closer to people—people like you, perhaps. Finding your doctor is one of the most important tasks your community can undertake. You stand to benefit not only from his professional services, but also from the contributions he'll make as a good citizen, from his participation in the life of the community."

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CONTINUED ON PAGE 62

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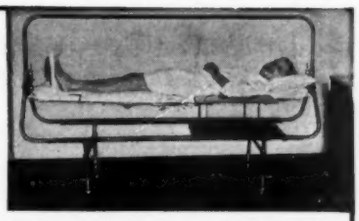

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NOTES AND QUOTES

Parke, Davis & Company, whose national advertising agency is Young & Rubicam, Inc., has been sponsoring "See Your Doctor" advertisements for the past twenty-six years.

Battlefield tests with a diagnostic agent called Evans blue dye revealed that the high fatality rates in epidemic hemorrhagic fever, a mysterious disease encountered by U. N. forces in Korea, were not caused by loss of blood as had first been assumed.

Two Army physicians, Drs. R. B. Giles and E. A. Langdon, who conducted extensive studies of the disease on Korean battlefields, report in the May, 1954, issue of The American Journal of Medicine *that this important new information about epidemic hemorrhagic fever was obtained by injecting patients with the blue dye and then measuring their blood volume by observing changes in the color of the blood.*

Epidemic hemorrhagic disease was first described by Russian doctors twenty years ago. The transmitting agent of the disease is still unknown, but evidence seems to point to rodent parasites such as fleas, mites, ticks and particularly chiggers.

More than 1,000 U. N. troops, mostly serving in forward areas, were hit by the disease in 1951, with fatalities ranging as high as 10 per cent.

THE treatment of cardiospasm with drugs, which they had previously found "discouraging," is now reported as "successful" by four physicians in Chicago who have employed a new "topical anesthetic-spasmolytic" preparation in three selected cases. Drs. H. Necheles, H. Laske, L. D. Elegant and R. Baum of the Department of Gastrointestinal Research of Michael Reese Hospital have had their study published in the May, 1954, issue of *The American Journal of Digestive Diseases.*

The preparation was an experimental liquid form of the drug called dactil. Chemically it belongs to the family of piperidols. Lakeside Laboratories, Inc. of Milwaukee recently made it available nationally in capsules to the profession.

"In previous experiments in animals and in man, we have demonstrated that a small dose of certain local anesthetic drugs applied locally to the intestinal mucosa, produced a greater local relaxation that the same or even larger doses injected intravenously," the authors explained.

"We have been encouraged in our present work by the appearance of a new drug which has both spasmolytic and topic anesthetic properties, and possesses such a degree of tolerance, that even large doses did not produce undesirable reactions," they continued.

The new drug, dactil, was dissolved in syrup "to maintain contact with the surface and prolong the local effect." The solution was administered before meals.

The American Heart Association and its affiliates have jointly bestowed 169 awards totaling $953,370.71 to scientists engaged in research in the field of heart and blood vessel diseases, it was announced by Dr. E. Cowles Andrus, president of the American Heart Association.

"These awards represent an increase of approximately $150,000 over the sum we were able to allot to the national program of research support last year," said Dr. Andrus. "They bring to nearly $8,000,000 the total the Association and its affiliates have provided for this purpose in the six years since it became a voluntary health agency. At the same time the number of applications worthy of support increased by nearly two-thirds. Many promising projects had to be denied support for lack of funds."

A UNIQUE program which provides scholarships in America and abroad to residents in training and plastic surgeons who have been in practice no longer than five years is meeting with considerable success, according to an announcement by Dr. Jacques W. Maliniac of New York City, chairman of the Board of Trustees of the Foundation of the American Society of Plastic and Reconstructive Surgery, Inc.

Now entering its sixth year of operation, the Foundation's program is attracting the attention of other medical and surgical groups which are interested in its system of providing scholarships on a reciprocal and "barter-type" basis. Its purposes are to improve practices of plastic surgery and to offer opportunities to men for wider training and worldwide contacts.

CONTINUED ON PAGE 64

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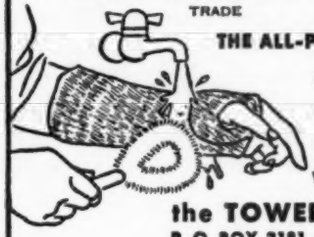

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NOTES AND QUOTES

The Foundation sponsors a yearly scholarship contest, Dr. Maliniac said, for well trained plastic and reconstructive surgeons in United States and foreign countries, outside the iron curtain. Two prizes, each consisting of a three-month plastic surgical scholarship and certificates of honorable mention, are offered this year. These give the winners the opportunity for assistantship in a great number of selected plastic surgical services here and abroad. The scholars are entitled to full maintenance in the hospitals.

The number of plastic centers made available to the scholarship pool in the United States and foreign countries has more than doubled since last year. As of April, 1954, there were twenty-five centers in the United States and Canada, two in Argentina, one in Brazil and one in Mexico. In Europe there were seven in the British Isles, one in Holland, three in Sweden and five in France.

An extensive program of clinical and laboratory research in the fields of vitamins and nutrition will be supported by eleven new grants to universities and medical centers throughout the country, it was announced by Dr. Robert S. Goodhart, scientific director of The National Vitamin Foundation. A total of $55,085.00 *to support the important studies has been granted by the Foundation for its broadened continuing program of scientific research. The National Vitamin Foundation gives grants-in-aid for research semi-annually throughout the United States and abroad. The new grants became effective June* 30, 1954.

MEDICAL treatment needed by young women with heart disease who want to have children should "logically begin in the period before marriage," according to Dr. Burton E. Hamilton, consultant in cardiology to the Boston Lying-in Hospital.

Since successful pregnancy for women with heart disease "depends in part on planned living and cooperation from the patient's family, timely education for all concerned is needed," Dr. Hamilton wrote in the June issue of *Circulation*, scientific journal of the American Heart Association. If surgery is indicated, for example, to repair damage to the heart valves caused by rheumatic fever or to correct congenital heart defects, it should preferably be undertaken in advance of pregnancy. Dr. Hamilton declares: "The need for surgery during pregnancy should be avoidable, except in rare circumstances, if cardiacs receive timely education."

The majority of young women with heart disease are not seriously handicapped by their condition and can expect to bear children successfully, Dr. Hamilton said. For such patients, whom he describes as "favorable" cases, the mortality rate goes up only slightly as a result of becoming pregnant. He emphasizes that this is true "only if the pregnant patient seeks and receives proper care." If not, she runs nearly as great a risk as if she were among the small minority of heart patients whose condition is poor.

To determine the effect of vitamin C on the healing of peptic ulcer the Florida Citrus Commission in Lakeland has made a grant to Dr. Frank C. ValDez, chairman of gastroenterology, Mercy Hospital, and associate clinical professor at Stritch School of Medicine, Loyola University, both of Chicago, Illinois. Blood and/or serum ascorbic acid levels will be followed throughout the period of study and treatment will be based on control of gastric acidity on a twenty-four-hour basis.

SOLVING the problem of deficit financing of the nation's medical schools by "billing" the graduate for his education after he is established in practice was suggested by Dr. Brian Bird in an article in the June issue of *The Journal of Medical Education*.

Dr. Bird, assistant professor of psychiatry at Western Reserve University School of Medicine, points out that for many years tuition charges have not been equal to the educational bill of medical students. Schools have met this situation by appeals for gifts from alumni, individuals, corporations, fund-raising organizations, charitable foundations and, reluctantly, government agencies.

If costs continue to rise or private sources decrease, Dr. Bird believes that the time would not be too distant when state schools would be the only ones able to pay their bills. This would

CONTINUED ON PAGE 66

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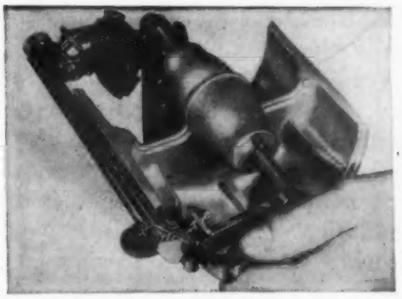

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mark the end of private medical schools, which many educators believe would be undesirable.

A way out of this dilemma, according to Dr. Bird, would be to regard the cost of medical school as a legal debt, which the student assumes with the understanding that he is to pay it back within a certain number of years after graduation. Dr. Bird feels it would work no hardship to ask them to pay in full for the education which enables them to earn so much. He says, "The doctor's role as a healer, a helper, a man of goodwill, must be preserved and can be preserved without cloaking him in a disguise."

Sulfa drugs combined with aureomycin makes it possible to treat infected abscesses, ulcers, bites, ingrown toenails, lacerations and other soft tissue infections with half the usual daily dosage of aureomycin alone, according to a study at Harlem Hospital reported in the June issue of the New York State Journal of Medicine.

Drs. James C. Dilorenzo, Louis T. Wright and William I. Metzger treated fifty-five patients, admitted to the hospital with fever for acute inflammations, with a daily dose of a half gm. of aureomycin and a little over a half gm. of sulfadiazine, sulfamethazine and sulfamerazine in combination tablets. They found that clinical results were equally as good as those obtained in an earlier study with a daily dose of 1 gm. of aureomycin used alone. They added that no signs of toxicity appeared which could be attributed to the use of the drug mixture.

EXPANSION of the Drug and Pharmaceutical Committee of the Committee of American Industry was announced by its chairman, Harry J. Loynd, president of Parke, Davis & Company. The Committee of American Industry, comprising sixty trade divisions, is an arm of the National Fund for Medical Education which has set an annual goal of $10,000,000 for the support of the nation's seventy-nine medical schools. Colby M. Chester, honorary chairman of General Foods, is chairman of the C.A.I.

Many corporation heads have agreed to join with him and other leaders in the drug and pharmaceutical industry to meet the division's goal for the National Fund.

"With the addition of these leading executives, our committee now comprises thirty heads of some of the largest firms in the drug and pharmaceutical industry," Mr. Loynd said. "Such splendid leadership augurs well for the contribution our industry will make to the maintaining of the country's medical schools as free, solvent and progressive institutions for turning out trained doctors sorely needed by industry, the hospitals and the whole community. These leaders have shown that the drug and pharmaceutical industry recognized its responsibilities in corporate citizenship, the obligation to carry a share of the load for human betterment."

Mr. Loynd stated that the drug and pharmaceutical industry last year contributed a total of $217,950 for distribution to the medical schools through the C.A.I. This was part of a total of $1,367,979 contributed by 994 corporations in 1953 and it was matched by $1,044,602 coming from practicing physicians throughout the country.

"Our industry's goal for 1954 is $750,000," Mr. Loynd said. "That is why the expansion of our trade committee is so important at this time. Much of the total will have been met by the end of June, in time for allotment to the medical schools to meet their budgetary requirements for the fall term. The balance of the 1954 contributions will accrue for distribution of grants at a later date."

Two Seattle physicians have reported that chances of complete recovery from meningitis are improved when patients are treated with terramycin® and sulfadiazine. Drs. William J. Warmington and Fred C. Moll write in The Journal of Pediatrics *that the therapy brought complete recovery to twenty-seven of thirty-one children suffering from the dread brain fever. Only two of the children died. (This mortality rate of 6.4 per cent may be compared with the rate of 50 per cent fatalities recorded during the meningitis epidemic of 1929, before the development of antibiotics and sulfas.)*

After administration of terramycin and sulfadiazine, both by injection, Drs. Warmington and Moll found, "The response to therapy was satisfactory in most patients. The acute neurological symptoms subsided promptly, oral feedings and medication could nearly always be given

CONTINUED ON PAGE 68

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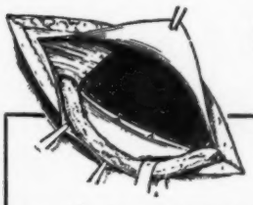

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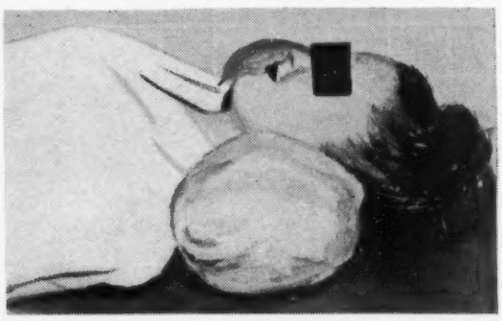

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after twenty-four hours and the fever returned to normal rapidly."

THE Tobacco Industry Research Committee invited university, hospital and other medical research organizations throughout the nation to submit proposals for specific cancer research projects for consideration by the Committee's Scientific Advisory Board.

At a meeting of the Committee's Scientific Advisory Board in New York, acting chairman Dr. Clarence Cook Little, director of the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine, said that the Board was in the process of reviewing sixty applications and inquiries already received.

An initial fund of $500,000 for research has been appropriated by the Tobacco Industry Research Committee which is composed of major cigarette manufacturers, tobacco growers and warehousemen. This committee has also pledged to vote substantial additional funds for specific projects upon the advice of the Scientific Advisory Board.

Composed of seven outstanding doctors, educators and scientists, the Advisory Board is guiding development of a long-term research program to be conducted in institutions outside the tobacco industry. In accordance with policy set when the program was announced early this year, the industry will not set up any research facilities. No industry funds will be given to tobacco company laboratories. All projects will be in addition to research carried on by companies.

A comprehensive program of clinical and laboratory research on disorders of the genitourinary system will be supported by a group of seven grants-in-aid to universities and medical centers throughout the country, it was announced by Dr. Thomas D. Moore of Memphis, Tennessee, president of the American Urological Research Foundation. This organization was formed and received its charter in April, 1952, and is devoted exclusively to research dealing with the problems relating to disorders involving the kidneys, bladder, prostate gland and genitalia. These include such common ailments as stone formation, prostatic obstruction in the aged, infections involving the urinary tract, such as pyelitis of pregnancy and pyelitis of infancy and childhood, Bright's disease, cancer involving the component parts of the genitourinary system and the problems of sterility in both sexes. The broad program of research instituted by the Foundation, as announced by Dr. Moore, consists of grants totaling nearly $35,000.00.

He concludes that the long established practice in diarrhea of starving infants or reducing their caloric intake "is based upon the false premise that the entire intestinal tract is affected by the disease." That this does not occur was demonstrated by the normal weight gains recorded by the group given a high calory diet. Thus, Dr. O'Keefe states, the "absorptive function is clearly unimpaired."

The high calory diet was fed orally to an unselected series of infants in private practice. The diet consisted of appella, boiled whole milk or evaporated milk and water in equal parts, cereal, strained beef or lamb, a multivitamin and liquid iron preparation. The net weight gain of this group reached "the amazing total of 351 ounces," the article notes. Appella is manufactured by Winthrop-Stearns Inc.

Thirty-six doctors from six foreign countries and thirteen states attended the fortieth annual session of the Trudeau School of Tuberculosis which ended its four-week course on June 25th.

Among the foreign countries represented were Canada, Australia (Tasmania), Thailand, Japan, Korea and Brazil. Some of the doctors were in private practice; others were public health officials, staff doctors at various hospitals and instructors in medicine in universities. Six were from the Department of Mental Hygiene of New York State, five from the Veterans Administration, four from the U. S. Navy, two from the U. S. Air Force and one each from the U. S. Public Health Service and the American Korean Foundation.

Sessions of the School were held at Trudeau Sanatorium and other divisions of the Trudeau-Saranac Institute, as well as Ray Brook State Tuberculosis Hospital, Sunmount Veterans Administration Hospital, Variety Clubs-Will Rogers Hospital, Sanatorium Gabriels, Stony Wold Sanatorium and the Saranac Lake Rehabilitation Guild.

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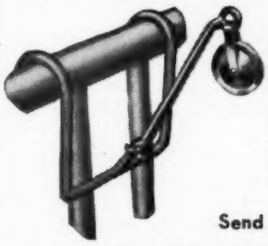

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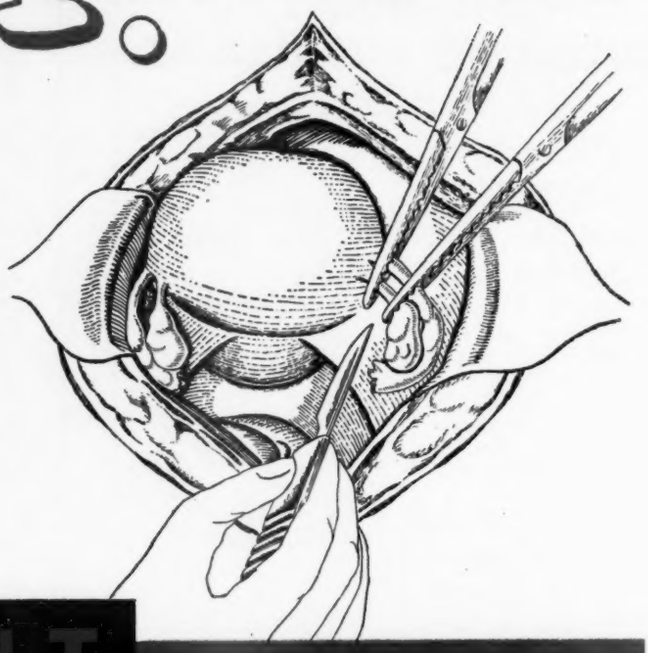

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THE American JOURNAL OF Surgery

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